COMPLICATIONS AND OUTCOME OF ACUTE ISCHEMIC STROKE

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CERTIFICATE

This is to certify that the dissertation entitled "**COMPLICATIONS AND OUTCOME OF ACUTE ISCHEMIC STROKE"** is a bonafide record of work done by **Dr.CHENNAPPAN.C** in the Institute of Neurology, Rajiv Gandhi Government General Hospital & **MADRAS MEDICAL COLLEGE, CHENNAI** in partial fulfillment of the Tamilnadu Dr.MGR Medical University rules and regulations for the award of **D.M. (NEUROLOGY)** degree under my direct guidance and supervision during the academic year **2011-2014**.

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DECLARATION

I solemnly declare that this dissertation titled **"COMPLICATIONS AND OUTCOME OF ACUTE ISCHEMIC STROKE"** is done by me in the Institute of Neurology, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai under the guidance and supervision of **Prof. Dr. K. BHANU, Dip. NB., D.M.,** Professor of Neurology, Institute of Neurology, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai. This dissertation is submitted to the Tamil Nadu Dr.MGR Medical University, Chennai in partial fulfillment of the university requirements for the award of the degree of D.M. Neurology.

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CONTENTS

INTRODUCTION

Stroke is an important cause of disability and second most common cause of death, worldwide. Developing countries account for two thirds of death due to stroke [1, 2]. As per the recent reports, the incidence and thirty day fatality rate is more in India when compared to developed countries[3]. The final outcome of stroke patients has been improved with various strategies like early diagnosis, early prophylactic treatment, early recognition of complications and mobilization[4].

Although the direct effect of brain damage is the main cause for immediate mortality after stroke, the early mortality that occur within the first month after an acute stroke is commonly due to preventable medical complications, that occur either early or late during the course of recovery from stroke.[5] These complications can be infections, neurologic-like recurrent stroke or seizures, thromboembolism, psychological problems, pain, and bed sores. High incidence of complications, ranging from 40% to 95%, following stroke have been found in previous studies.[6,7,8,9] There have been many limitations in the previous studies of complications of acute stroke. Individual complications have been concentrated in

most published studies. Different diagnostic criteria were also used in these studies to classify the complications.

A multicenter study (CAST-1) was conducted to examine the frequency and the factors associated with complications in acute stroke, as well as complications that could influence the outcome.[10] Identifying the complications will aid the development of preventive strategies aimed at improving outcome after stroke.

REVIEW OF LITERATURE

NEUROLOGICAL COMPLICATIONS

Although advances have been made over the past two decades in the diagnosis and management of acute stroke, there is still high mortality after stroke which makes it the common cause of death in the developed world, after ischemic heart disease [11]. 23-50% of deaths in patients with ischemic stroke are caused by the post stroke complications[12]. These complications, even when not life

threatening, can prolong hospital stay, rehabilitation, functionally poor outcome and increased cost of care[12 - 14]. Both neurological and medical complications are found to occur following ischemic stroke. Recurrent stroke, brain oedema, hemorrhagic transformations, seizures and delirium are the neurological complications.[12,15,16] Though these complications occur earlier- 48 to 72 hours of stroke onset, they are less frequent than medical complications.[15] Some previous studies have indicated that direct consequence of neurological complications like brain damage to cause death after stroke in the first few days.[17,18]

Brain oedema

One of the major causes of death within the first week of stroke is brain oedema.[19]Energy depletion in cerebral ischemia leads to ionic imbalance and causes brain oedema. Ischemic stroke patients are found to have two types of oedema- vasogenic and cytotoxic oedema. Characteristic feature of cytotoxic

oedema is expansion of intracellular compartment by translocation of water from interstitium, with intact blood brain barrier.[20] Blood brain barrier is compromised at the late stage of stroke causing vasogenic oedema which is characterised by fluid movement into extra vascular phases from the vascular compartment. There is expansion of brain volume due to vasogenic oedema which causes an increase in intracranial pressure, brain herniations and other ischemic injuries.[21] Since cytotoxic oedema doesn't respond to anti- oedema treatment, the differentiation between vasogenic and cytotoxic oedema is important both for diagnostic and therapeutic purposes. This is done with the help of MRI: Vasogenic oedema causes water retention in brain tissues which can be seen on fluid –attenuated inversion recovery images and conventional T2- weighted images. On the other hand, there is overall reduction in the diffusivity of water molecules in cytotoxic oedema and is seen as high signal intensity in diffusion weighted images. Age of the patients as well as the extent and location of the infracted area determines the extent of swelling. Malignant middle cerebral artery syndrome and fatal brain oedema tend to develop more in younger patients than their older counterpart.[22] Older people are protected from developing space- occupying brain swelling probably because of cerebral atrophy, as shown in animal studies where significantly less stroke- induced oedema is seen to develop in ageing mice than in young animals.[19]

Hemispheric oedema

There is 10-20% risk of cerebral oedema in patients with ischemic stroke involving the anterior circulation.[23] Patients who have major anterior circulation occlusion tend to develop cerebral oedema within 4 days after stroke. Patients with large cerebral infarctions along with brain oedema usually present in coma. Brain oedema with midline structure shift is compression of brain stem is an important cause of mortality.[24] Complete infarction of MCA territory and herniation of brain within 20 hours of onset of symptoms leads to condition called malignant MCA infarction.[19] This kind of infarction is among the most devastating and life threatening neurological complications of ischemic stroke. Malignant MCA infarction accounts for a mortality rate between 40% and 80%, whereas that due to acute MCA infarctions caused by brain oedema and cerebral herniation is between 7% and 23%. Development of malignant MCA infarction can be predicted with a high sensitivity (91%) and specificity (94%), using Ct perfusion imaging which shows large areas of hypo perfusion and enhanced CT showing large hypo attenuation.[25] Other predictive imaging findings include severe perfusion deficits on perfusion-weighted MRI within 6 hours or single PET scan, large diffusion- weighted imaging lesion volume and the decrease in diffusion coefficient in large area within 6 hours of stroke.

Management

Elevation of the head end to 20-30 degree angle is used in the initial management of increased intracranial pressure, after an acute ischemic stroke, as it improves the venous drainage. Some of the other factors like hypoxia, hyperthermia, hpercapnia, hyperglycaemia and antihypertensive drugs, which can increase intracranial pressure, are to be avoided. Hemicraniectomy is recommended in selected patients who have substantial brain ischemic swellings and brain shifts that are life threatening.[26] The principle behind removing the part of cranium is to prevent secondary damage to brain tissue and improves collateral perfusion by creating enough space for the expanding brain.

Haemorrhagic transformation

A potentially serious and common complication of acute ischemic stroke is haemorrhagic transformation of brain infarct which occurs in 30-40% of clinical cases. Disruptions of neurovascular homeostasis and micro vascular integrity are the major causes of haemorrhagic transformation. There is an increased frequency of haemorrhagic transformation in patients managed with mechanical embolectomy and intra-arterial fibrinolytics (7%), intravenous alteplase (6%) than in patients who are treated with supportive care alone (0.6%).[27] The risk of serious haemorrhagic transformation is increased with the use of anti- thrombotic, especially anti coagulants, in addition to thrombolytic drugs, after ischemic stroke. The risk of clinically detectable haemorrhage is increased when aspirin is

used early. There is more possibility of development of haemorrhagic transformation in elderly patients with stroke due to factors which include impaired clearance of alteplase, age associated micro angiopathy, and increased frequency of transformation in cardio embolic infarcts when compared to atherosclerotic infarct.[28]

NINDS system classifies haemorrhagic transformation into two types- intra cerebral haematoma and haemorrhagic cerebral infarction. Intra cerebral haematoma is defined as CT findings of a homogenous, hyper dense lesion with or without oedema or mass effect within the brain. CT findings of an acute infarction with an irregular border within the vascular territory and with variable hyper and hypo density define haemorrhagic cerebral infarction. Haemorrhagic transformation expands the brain oedema leading to apoptotic glial cell and neuronal cell death, increased intracranial pressures, disruption and displacement of brain structures with high rate of mortality.[29]

Clinical state of the patient, risk of subsequent arterial or venous thromboembolism and the risk of recurrent intracerebral haemorrhage determines whether or when to restart anti thrombotic therapy after haemorrhagic transformation. Antiplatelet drugs might be a safer choice when compared to warfarin for patients with a high risk of re-bleeding but relatively lower risk for cerebral infarction; conversely, patients with a very high risk of

thromboembolism can be restarted with warfarin after 7-10 days of onset of intracerebral haemorrhage.[30]

Seizures and epilepsy

Seizures after ischemic stroke can either occur soon or can be delayed. Early seizures are defined as those which occur within 1 or 2 weeks of stroke and late seizures are those which occur thereafter. The frequency ranges from 2% to 23% for early seizures and between 3% and 67% for late seizures, depending on the study design, sample size and length of follow-up. Epilepsy (recurrent seizure) is seen to develop in only 2.5% to 4% of patients.

Cellular biochemical dysfunction causing electrically excitable tissue results in early seizure after stroke whereas development of meningocerebral gliosis and cicatrices are thought to result in late seizures. Several risk factors which have been identified includes stroke severity, multiple site involvement, embolic stroke, large cortical infarcts, size of the infarct, decreased consciousness and metabolic and hemodynamic disturbances.[31, 32]

Seizures are found to occur more often in patients with cranial sinus thrombosis compared to those with arterial stroke. It may be the initial clinical presentation in central venous sinus thrombosis.

In some cases clinically undetectable non-convulsive seizures might account for deteriorating function. Recurrence rate is 50% in patients with late onset seizures whereas it is only 16% with early onset seizures. Frequency of recurrent seizures is related to the infarct and associated neuronal death. With the recurrence of late onset seizures, there is promotion of vascular cognitive impairment and the disability of patients with stroke is increased. In one study with 1220 patients, the in- hospital mortality rate in patients without seizure was only 14.4% compared to 37.9% in patients with early seizures. Conversely in two other studies, early seizures were associated with better outcome with regard to Scandinavian stroke scale scores.

It is a common practice to start short-term antiepileptic drug treatment for 3-6 months to treat recurrent early seizures, whereas for late seizures long-term conventional therapy is needed. However, no study has assessed the advantages and disadvantages of short-term and long-term therapy. There is no need for long-term antiepileptic to prevent of recurrence of early seizures. When compared to late onset seizures, in a retrospective study which particularly examined the risk factors for developing epilepsy.[35] One study showed patients with first, late post-stroke seizure was treated with Gabapentin monotherapy found to be 80% seizure remission after 30 months.[36] Status epilepticus and early onset seizures are treated with intravenous benzodiazepines as the first choice drug, eventually

followed by sodium valproate, phenytoin, gabapentin or lamotrigine are the first-line treatment for post-stroke seizures and epilepsy in younger patients who need anticoagulants or in elderly patients, and carbamazepine for patients who need no anticoagulation and with no bone health problems.[37]

There is no recommended usage for anti convulsants as prophylaxis in patients with any seizures following a recent stroke.[26]

Recurrent stroke

The risk of recurrent stroke, after being high in the first week in patients with acute ischemic stroke, declines over time. Risk of recurrence is about 10%, between 2%-4% and about 5% at one week, one month and then yearly, respectively. The major risk factors for recurrent stroke include previous stroke, old age, hypertension, diabetes mellitus, cardiac diseases, atrial fibrillation, carotid stenosis, and smoking.[38] Data from some studies indicate that patients with large artery atherosclerosis have the highest risk for stroke recurrence when compared to other etiological factors. Patients at risk of early recurrence stroke can be identified using transcranial Doppler which detects micro embolic signals. Prognostic score given by integration of clinical and imaging information score (recurrence risk estimator at 90 days [RRE- 90 score]) is used in the prediction of

risk of early recurrence after ischemic stroke and could help to improve the clinical practice in acute stroke care.[39]

Each additional recurrence stroke increases the risk of early and late mortality, severe disability and dependency. Early clinical deterioration due to recurrent stroke was seen in 4.5% of 8291 patients with minor or transient ischemic stroke whereas it was 11.3% in 1964 patients who had a stroke.[40]

Management

The need for early secondary prevention is substantiated by the early increased risk of recurrent stroke. Therefore, it is imperative to identify the cause and treat the stroke when possible. There is good evidence that early mobilisation and correction of abnormal physiological variables which will improve the clinical outcome and reduce the risk of recurrence of stroke.[26] Multifactorial approach involving the blood sugar control, antiplatelet therapy, treatment of hypertension, reduction of elevated cholesterol, carotid endarterectomy, lifestyle changes and anticoagulation for atrial fibrillation might prevent at least 95% of recurrent strokes.[41] Patients with ischemic stroke and symptomatic atherosclerotic narrowing of large intracranial or extracranial arteries have the options of surgical and endovascular interventions for treatment.

Delirium

Delirium is characterised by acute transient disturbance of consciousness and a change in cognition with fluctuating quality. The prevalence of delirium ranges from 13% to 48% among the patients with acute stroke. Some studies showed 50% of the patients have delirium after one week after stroke. Though the cause of delirium in stroke is poorly understood, changes in the neurotransmitter concentration (serotonin, norepinephrine, acetylcholine, GABA and dopamine), hypothalamic- pituitary- adrenal axis activation, non-specific reaction to stress might have a role.[42] Post-stroke delirium may be due to hypoperfusion in the frontal, pontine and parietal regions as indicated by single photon emission CT scans. Also in another study, there was an association between delirium and hypercortisolism in acute stroke. Risk factors for the development of post stroke delirium are pre-existing cognitive impairment, in previous stroke, poor pre-stroke vision, old age, acute confusional states, apnoea, sleep apnoea, earlier treatment with anti-cholinergic drugs, left- sided brain lesions, total anterior circulation infarction, lesions in the caudate nucleus and thalamus, dysphagia on admission, intracerebral haemorrhages, cardioembolic stroke, neglect, infectious and metabolic disorders have all been identified. Post stroke delirium increases

the risk of dementia, increases admission to an institution and prolongs hospital stay.[43]

Management

Treatment of post stroke delirium is usually the same as delirium in patients with other diseases.

Central post- stroke pain

It occurs after subcortical, capsular, ventroposterolateral thalamic infarcts and lower brain stem infarcts, lateral medullary infarcts and anterior spinal artery infarcts. It is also known as *dejerine roussy syndrome* or thalamic pain syndrome. The infarcts in patients with central post-stroke pain are characterized by involvement of the spinothalamic tract with sparing of the lemniscal pathways, as evidenced by normal somatosensory evoked potentials.[44] The prevalence of central post-stroke pain ranges between 1%-12% in all patients with stroke and 18% of patients with a somatosensory disturbance. The onset of the symptoms to develop may take from days to years, but usually occur after several months. It will interfere with sleep and rehabilitation. Amitriptlyline and lamotrigine are the

first line drugs and the second line drugs are gabapentin, fluvoxamine and mexiletine. Propofol and lidocaine can be given for short term pain relief.

Headache

Acute ischemic stroke is usually accompanied by headache, occurring before (43%-60%; sentinel headache), concurrently (25%-30%; onset headache), or after (14%-27%; late- onset headache) focal neurological deficit. Headache following acute stroke normally begins on the first day. It is usually severe and continuous in nature and is of pressure type, lasts for 3-8 days. In the aftermath of major strokes, headaches are very common. Patient with anterior circulation have lesser chance of headache than patients with vertebrobasilar ischemia, because vessels are more densely innervated by nociceptive afferents in the posterior circulation than are those in the anterior circulation.[45] In one study with onset headache, early neurological deterioration could be strongly predicted (98% of positive predictive value, 56% sensitivity and 99% of specificity).[46] Secondary stroke prevention treatment drugs like dipyridamole can cause headache. Delayed headache are caused by factors like haemorrhagic transformation, oedema, intracranial hypertension, delayed interruption to the function of trigemino vascular system and delayed effects of ischemia and thrombosis by-products.

Post stroke headache respond to analgesic like paracetamol or might recover spontaneously. Opioid drugs have side effects like respiratory depression and hypotension and also alter the clinical picture and hence should be avoided.

MEDICAL COMPLICATIONS

Deep vein thrombosis and pulmonary embolism

One of the lethal and most feared complications in stroke patient is pulmonary embolism. Though it occurs in only 1% of stroke patients, pulmonary embolism accounts for 15% of deaths.[47] Deep vein thrombosis of lower extremities is found in a great majority of patients who develop pulmonary embolism. Patients who are yet to walk tend to develop thrombi mostly in their paretic limb whereas some venous thrombi are found in the pelvic structures. The main causative factors that promote venous thrombosis and thromboembolism are an increase in acute phase reactants following brain ischemia which increases blood coagulability and stasis of blood.

Pulmonary embolism, a life- threatening complication for patients with stroke is preceded by a serious problem of deep vein thrombosis. After an acute stroke there is 2 to 10 percent prevalence of clinically evident DVT and even a higher prevalence of asymptomatic DVT.[48] 18 percent of patients with acute stroke were found to have proximal DVT at 21 days in a study which used magnetic resonance imaging (MRI), and 28 to 80 percent patients were detected to have occult DVT in a study that employed fibrinogen scanning in patients with acute stroke and hemiplegia. [49]

DVT may develop as early as the second day with a peak incidence between two and seven days.[50] There is 15% risk of death in patients with stroke with an untreated proximal DVT.[50]There is a predisposition to development of DVT in patients with hemiparesis and the degree of paresis is directly proportional to the risk of DVT. There may be 75 percent risk of DVT in the paretic side and on the nonparetic side it is 7 percent.[51] High stroke severity, advanced age, atrial fibrillation and immobility are other additional risk factors for DVT. The noninvasive test of choice for patients with suspected DVT is compression ultrasonography.

Patients who happen to harbor deep vein thrombosis detected by venography and noninvasive techniques often have no abnormal symptoms or physical signs. Deep vein thrombosis is more likely to develop in patients with congestive heart

failure and atrial fibrillation and those with severe leg weakness.[52] In patients with hemiparesis the paretic limbs are most affected with venous occlusions. Since most of the pulmonary emboli are silent their true frequency is not known.

Use of subcutaneous LMWH or low- dose unfractionated heparin in order to prevent DVT and PE in patients with acute ischemic stroke with restricted mobility is recommended by most evidence-based clinical guidelines.[53] Either subcutaneous heparin 5000U twice or thrice daily or LMWH like enoxaparin 40mg daily can be used to prevent VTE in patients with acute ischemic stroke with restricted mobility. Patients with contraindications to anticoagulants are recommended intermittent pneumatic compression.

Patients with sudden chest pain, shortness of breath, change in respiratory pattern, hemoptysis, hypotension, hypoxemia, confusion, agitation are suspected to have pulmonary embolism. Evaluation should begin depending on the index of suspicion. Tests included are the following:

1. Chest x-ray

2. Electrocardiogram

3. Arterial blood gas analysis

4. Noninvasive studies of lower limb venous circulation

5. Nuclear lung ventilation and perfusion scan

6. Dye contrast angiography or pulmonary CT angiography

Urgent treatment with intravenous full-dose heparin or LMWH is used in patients who are found to develop VTE or pulmonary embolism.

Cardiac abnormalities

Mortality due to cardiac abnormalities is the second most common cause of death in patients with acute stroke, next only to neurological complications. Cardiac related death is highest during the first month after stroke and thereafter it declines in frequency.

Stroke patients even in whom there is no previous heart disease are found to have electrocardiogram changes consistent with ischemia, elevated troponin levels, creatine-phosphokinase-myoglobin levels and various cardiac arrhythmias. [54] Cardiac abnormalities are most likely due to lesions of particular regions of the brain. There are three ways by which stroke causes secondary cardiovascular, cardiac and respiratory changes. They are as follows,[55]

1.Critical structures such as the brainstem nuclei, hypothalamus and the cortex of the Insula of Reil that make up the central autonomic network, which activate autonomic descending fiber pathways to the lungs, heart and blood vessels are directly involved.

2. Compression of the brainstem or hypothalamus, or both has a mass effect which activates the autonomic pathways

3. Stress effects caused by acute brain lesion trigger the release of catecholamines and corticosteroids by stimulating the hypothalamic-pituitary axis.

Cardiovascular effects like arrhythmias are seen in strokes that involve the insular cortex .Acute non-lacunar middle cerebral artery territory infarcts are the ones which commonly involve the insular cortex. Direct involvement of the medulla oblongata and brainstem compression can lead to vagal discharges, which can cause cardiac arrythmias, sinus bradycardia and even cardiac arrest, as well as a fall in diastolic blood pressure and elevation of systolic blood pressure changes found in patients with brain herniations and increased ICP usually called the Cushing response.

An increase in sympathetic tone is caused by stroke which elevates the levels of catecholamines that cause focal myocardial damage and subsequent arrhythmias. Patients with pre existent ischemic heart disease is especially more

prone for myocardial infarction following a stroke. Coronary thrombosis is promoted by an increase in acute phase reactants that follows a stroke. The highest risk for cardiac arrhythmias is seen in older patients with cerebral hemispheric infarction.[56] Patients with medullary and cerebral hemispheric infarcts commonly have heart rate variability. Patients with neurogenic cardiac arrhythmias can be treated both for arrhythmias and its cause using propranolol or other beta blockers.[56]

Swallowing Abnormalities

Dysphagia, defined by swallowing impairment of the upper digestive tract, is more specifically characterized as oropharyngeal dysphagia when related to stroke. The extended definition captures impairments in swallowing efficiency and safety that includes reduced range of movements, delay in the timing of movements and frank aspiration. [57] Dysphagia which is a major risk factor for the development of aspiration pneumonia is very common after stroke. Systematic review of 24 studies which evaluated oropharyngeal dysphagia and aspiration in patients with stroke support the above mentioned observations.[57]

The incidence of dysphagia after stroke was lowest, intermediate and highest when identified by screening methods like water swallow (37 to 45 percent),

trained swallowing (51to 55 percent) ,and instrumental testing, mainly video fluoroscopy (64 to 78 percent), respectively.[57] The dysphagia rates identified by instrumental testing may have been overestimated because the movement patterns due to the effect of aging were not distinguished from pathological dysphagia as per the definitions used in the studies.

On initial presentation, age more than 70, male gender, incomplete oral clearance, impaired pharyngeal response, disabling stroke and palatal asymmetry or weakness are the independent predictors of dysphagia.

Screening bedside tests for swallowing dysfunction though are important they are not accurate enough. Overall sense of the presence of aspiration by the examiner and spontaneous cough during test swallows were found to be the best bedside predictors of aspiration to thin liquid in one study.[58] When a formal screening protocol for dysphagia like water swallow test was used in a prospective, multicentre study, for all stroke patients, it was found to be associated with a significantly decreased risk of aspiration pneumonia when compared with no formal screen.[59]

Approximately one fourth to one third of patients with stroke is found to have symptomatic dysphagia. Patients who have had strokes that involve the brainstem or bilateral hemispheric strokes are more commonly seen to have dysphagia and

aspiration.[60] It is also common in patients with severe strokes and in those with reduced consciousness and dementia.

Predictors of increased risk of aspiration are dysarthria, dysphonia, voice change after swallowing and abnormal cough to extricate food from the oropharynx. A potential risk for aspiration can also be detected by a change in pulse oximetry following swallowing.[61] Abnormalities of swallowing can be detected in approximately 50% of stroke patients using video fluoroscopic studies.

Choice of foods, instructions to the patients, physical therapy with guidance in positioning of food within the oral cavity and pharynx and the use of thermal stimulation can help prevent aspiration. Although there is temporary need for feeding tubes most of the patients resume feeding within months.

Pulmonary Complications

One of the most common complications occurring in about 5 percent of acute stroke patients is pneumonia.[6] In patients admitted to a neurologic intensive care unit with acute ischemic stroke and in who require nasogastric tube feeding there appears to be a higher incidence of stroke-related pneumonia(21 and 44 percent, respectively).[62]

Pneumonia is the most common medical complication within four weeks of a supratentorial ischemic infarction and the most common cause of fever in the first 48 hours of an acute stroke.[6] Also most of the hospital readmission in stroke survivors in the first five years following an ischemic stroke are due to respiratory illness and pneumonia, as suggested by a retrospective data. An abnormal water swallow test, age more than 65 years, cognitive impairment, severe poststroke disability and dysarthria or dysphasia are independent risk factors for pneumonia, in a prospective study of 412 patients with acute stroke.[63] Decreased level of consciousness and facial palsy were independent risk factors for pneumonia , in patients who needed nasogastric feeding. About 60 percent of post-stroke pneumonia is caused by aspiration.[6] Abnormal entry of fluid, endogenous secretions or exogenous particulate substances into the lower airways and its pulmonary consequences is known as Aspiration pneumonia. Aspiration of microorganisms from the nasopharynx or oral cavity is the common cause for most pneumonia.

After stroke, pneumonia is common in the immediate and late periods. Pneumonia was a complication in 33% of patients dying with cerebrovascular disease, in a retrospective post-mortem study.[64] Poor mobilisation of secretions and atelectasis often occur in a recumbent position. Aspiration may be caused by difficulty in swallowing. There may be no or only poorly performed coughing and

deep breathing. Also there are decreased chest movements on the hemiplegic side. Pneumonia and other infections, both in the hospital and afterwards, can also develop due to an immuno depressed state created by the stroke.[65] Patients should be tested for swallowing function before oral feeding. Pneumonia after stroke can probably be prevented by physiotherapy or nursing technique.

Metabolic and Nutritional Disorders

An important but seldom recognized complication of stroke, especially in elderly patients whose nutritional intake was poor even before their stroke, is prolonged under nutrition. It also serves as a predictor of poor outcome. Good gains in neurological functional status was seen when the serum albumin levels were high.[66] Abnormal bone metabolism, gastrointestinal and cardiac dysfunction and diminished immune functions can result from malnutrition. Malnutrition also contributes to the formation and repair of decubitus ulcers.

Gastric feeding tubes may be required when there is prolonged inability to swallow. Percutaneous endoscopic gastrostomy (PEG) tubes have helped many stroke patients maintain nutritional balance in spite of dysphagia. The outcome in stroke patients can improve with early PEG placement. Nutritional, electrolyte and fluid abnormalities can also occur during the acute stroke and recovery

periods. Hyponatremia develops in approximately 15% of acute stroke patients. Inappropriate secretion of antidiuretic hormone (ADH) is responsible for post stroke hyponatremia as posited by Joynt et al.[67]

Some of the potential mechanisms, like increased release of ADH, damage to a widespread vasopressin neuronal system, resetting of osmoreceptors, effects on ADH secretion related to recumbency, damage to the anterior hypothalamus, and secondary stroke- related elevations in serum catecholamines and cortisol were reviewed by Joynt et al.[67] Abnormalities of serum sodium concentrations have been posited to be caused by changes in the levels of atrial natriuretic factor. Volume depletion and overload must be excluded as contributing factors in hyponatremic patients.

Urinary Tract Infections and Urinary Incontinence

One another common complication of stroke is urinary tract infections. Out of 1455 patients, 17.2% developed urinary infections in an international trial of a neuroprotectant.[68] The following two factors are probably the cause for a high frequency of urinary tract infections. The first is the placement of an indwelling catheter to empty the urinary bladder. This serves as a source for introduction and growth of bacteria. Hence continuous bladder drainage with indwelling catheter

should be avoided. Using strict sterile techniques, intermittent catheterization is preferable. Condom catheters suffice in some men. Second, stroke can alter the functioning of the external sphincter and urinary bladder. Urinary symptoms like frequency, urgency and retention are common even with uninfected bladders. Obstructive uropathy due to large prostrate is seen in men in the stroke age group who are often geriatric.

Gastrointestinal Bleeding

Some patients with stroke and other brain diseases developing gastrointestinal haemorrhage has long been realized by physicians. This problem is usually called stress ulcers, *Cushing's ulcers*, or hemorrhagic gastritis and when severe, can be life threatening. At a hospital in Edinburg, 18 of 607 (3%) patients with stroke developed gastrointestinal hemorrhages, as reported by Davenport and colleagues. One half of the hemorrhages were severe.[69] Most patients had melena or hematemesis, but one patient suddenly developed abdominal pain and went into hemodynamic shock.[69] Gastrointestinal bleeding most commonly develops in older patients with decreased levels of consciousness and severe stroke. Risk of stress ulcers is also increased by corticosteroids that are given for brain oedema.

H2 antagonists are recommended for prophylactic use in patients with large strokes and reduced awareness.

Pressure Decubitus Ulcers

Bedsore is an iatrogenic complication of stroke which is preventable and significantly hinders the rehabilitation process. Patients who are immobilized, do not sense the need to change position and whose ability to reposition themselves is restricted, are at risk of developing bedsores if they are not frequently repositioned.[70] The risk of developing skin breakdown also increases when there is incontinence.The patient should be turned frequently and the skin should be kept dry and clean. Nutrition should be given adequately. Pressure on immobilized or anesthetic limbs should be avoided. Heels can be spared from ulcers with the use of padded heel boots. Development of sacral pressure sores can be retarded with the use of waterbeds, egg-crate mattress or soft cotton padding.

The entire skin surface should be periodically examined by the physicians and nursing personnel for any area of early breakdown. Susceptible areas like the sacrum, buttocks, elbows, heels, toes, wrists and occiput should be paid particular attention. Pressure should be totally avoided on that area where an ulcer develops,

the wound should be dressed, special mattresses should be used and the skin debrided and grafted, if necessary.

Contractures and Shoulder Pain

Limb immobility when usually maintained in fixed, flexed positions at the knees and elbows can lead to fixed contractures. Frozen shoulders, shoulder pain, and the so-called shoulder-hand syndrome can develop with decreased shoulder movement. Out of 300 patients, 22% developed significant shoulder pain within 4 months following stroke, in the Lund Stroke Register.[71] The shoulder-hand syndrome was found to develop in 36 of 132 (27%) of patients with hemiplegic stroke, in one study.[112] Characteristics of the shoulder-hand syndrome includes, edema of the distal hand joints; swelling and pain over the carpal bones; pain and tenderness when flexing, abducting and externally rotating the upper arm.The likelihood of developing swelling of the upper extremity and shoulder pain is increased with severe shoulder spasticity, weakness and subluxation of the shoulder.Prevention of this disabling and unpleasant complication of stroke needs the shoulder joint to have an early full range of movement.

Low-dose corticosteroids and NSAIDS like indomethacin may be used in patients with shoulder pain. Another complication of hemiparesis is subluxation of shoulder. The weak arm should be well supported.

Peripheral Nerve Injuries

The most commonly involved nerve is that of peroneal nerve whose compression causes a foot drop. Compression of the nerve at the elbow, especially in wheelchair-bound patients, causes ulnar palsy. Occasionally, patients with reduced consciousness compress their femoral nerve in relation to pressure on the groin region.

Osteopenia and Osteoporosis

Significant reduction in bone mineral density was found to occur on the hemiplegic side when the bone mineral densities were studied after stroke. The multiple causes include poor nutrition with inadequate vitamin D stores; lack of sunlight exposure; immobilization-induced calcium resorption from bone; and osteoporosis before the stroke. In patients with severe hemiplegic and prolonged immobilization, the hemi osteoporosis is most severe.

Hip and other fractures tend to occur predominantly on the hemiplegic side due to reduced bone density.Patients who are at risk of this complication can be given vitamin D supplements and calcium prophylactically.Exposure to sunlight and early mobilization are also important. In one small study, Zoledronate when given, as a single intravenous infusion of 4 mg within 35 days after stroke, prevented hip osteopenia, in one small study.[73]

Depression

Patients with a history of depression prior to their stroke are prone to become depressed even after a stroke. Both major and minor depressive symptoms have been found to increase in prevalence, from 23% and 20%, respectively, immediately after the stroke to 35% and 26% at 6 months.[74] Since stroke related organic behavioral changes like apathy, abulia, impersistence, aprosodia and anosognosia are hard to separate from functional psychogenic reactions, there is often difficulty in the diagnosis. Because of slowed responses or aphasia, an accurate history may not be available. It may be difficult to interpret vegetative and autonomic signs also. There is often coexistence of psychological and neurologic effects which augment each other. Depressive patients become sad due to loss of function and cannot perform up to their potential. Left- hemispheric

stroke was associated more with depression than right-hemisphere strokes. Higher frequency of depression are correlated to the more anteriorly placed infarcts, in the left hemisphere.[75]

Constipation

Constipation is frequent in patients admitted with acute stroke. Several explanations for this may include: the patients are dehydrated, elderly, immobile and often treated with a number of drugs. Constipation is often caused by immobility and bed rest, which may in addition induce deconditioning, resulting in inadequate force to defecate. Hypovolemia is a frequent problem, both before and after stroke, the latter due to impaired thirst mechanisms, dysphagia and lack of attention to drinking possibilities. There is often an insufficient intake of dietary fibre by the hemiplegic population. The coordination between peristaltic wave and the relaxation of the pelvic floor and external sphincter is impaired when the sequencing of sympathetic and parasympathetic components of defecation is disrupted by lesions involving the pontine defecatory centre. [76]

AIM OF THE STUDY

Study the frequency of complications after an acute ischemic stroke and their impact on the outcome of patients.

OBJECTIVE

Whether any of the complications have an influence over the final outcome of the patient.

INCLUSION CRITERIA

- First ever stroke
- Ischemic stroke

EXCLUSION CRITERIA

- Patients who have hemorrhagic stroke
- Previous history of stroke.
- Patients with traumatic brain injury.

PLACE	: Rajiv Gandhi Govt General Hospital Chennai - 600003
STUDY DESIGN	: Prospective study
STUDY PERIOD	: December 2012 to December 2013
ETHICAL CLEARANCE	: Obtained
CONSENT	: Informed consent from all patients

MATERIALS & METHODS

100 Patients with first ever stroke who were admitted in Rajiv Gandhi Government General Hospital were recruited in the study. Informed written consent was obtained from the patients before their enrollment. This was a prospective study approved by the Institutional ethical committee.

Detailed history including age, sex, symptoms at the time of stroke and risk factors was collected. All patients were clinically examined and subjected to investigation like complete blood count, renal function test, serum electrolytes, lipid profile, serum protein,ECG,ECHO,CT brain, carotid and vertebral Doppler and MRI brain in selected patients.

Stroke was classified into anterior circulation stroke and posterior circulation stroke. Severity of was classified using the National Institutes for Health Stroke

Scale (NIHSS) as mild to moderate (score ≤10) and moderately severe to severe (score ≥11).

Occurrence of complication was observed in all patients during their hospital stay. Complications were defined according to standard criteria [7](table 1) and included infections (UTI,RTI),shoulder pain, thromboembolism (DVT and Pulmonary embolism),complications of immobility (pressure / bed sores),psychological(depression) ,gastrointestinal (constipation,UGI bleed) and neurological (seizure,haemorrhagic transformation, brain edema).

Complications, when present, were treated accordingly. Patients were observed during their hospital stay, at the time of discharge and after one month. Assessment of functional outcome was done using the modified Rankin Scale (mRS). Patients who were not able to attend the follow -up clinic were assessed through phone calls. Outcome was grouped as good (mRS score 0-2) or poor (mRS score 3-6).

Table 1-Definition of complications

Infections

UTI: Clinical symptoms of UTI or positive urine culture

Chest infection: Auscultation of respiratory crackles and fever or radiographic evidence, or new purulent sputum

Other infection: Any pyrexia illness lasting for24 Hours

Pain

Shoulder pain: Pain in the shoulder area requiring analgesia on 2 or more consecutive days

Other pain: Any other source of pain requiring regular Analgesia

Thromboembolism

DVT: Clinical diagnosis of DVT

Pulmonary embolism: Clinical diagnosis of pulmonary Embolism

Complications of immobility

Falls: Any documented falls regardless of cause; fall with serious injury defined as

one resulting in fracture, radiologic investigation, neurologic investigation, or wound suturing

Pressure sore/skin break: Any skin break or necrosis resulting from either pressure or trivial trauma (skin trauma directly resulting from falls included)

Psychological

Depression: Low mood considered to interfere with daily activities or require pharmacologic or psychiatric intervention

Emotionalism: Episodes of crying or laughing that are sudden or unheralded and not under social control

Anxiety: Symptoms of anxiety considered to interfere with daily activities or requiring pharmacologic or psychiatric intervention

Confusion: Cognitive disturbance considered to interfere with nursing care or rehabilitation

Neurologic

Recurrent stroke: Clinical features lasting more than 24 hours consistent with the World Health Organizations definition of stroke. The new event is unequivocally in

a different arterial territory from an earlier one during the hospital stay.

Epileptic seizure: Clinical diagnosis of focal and/or generalized seizure in a previously nonepileptic patient

Unexplained events

Miscellaneous: Any documented complication resulting in a specific medical or surgical intervention (eg, gastrointestinal hemorrhage, constipation, episodes of cardiac failure, cardiac arrhythmias, arthritis)

RESULTS AND ANALYSIS

This study commenced with 100 patients of acute ischemic stroke who were followed up for 1 month from the onset of stroke. Various complications during their hospital stay were noted and treated accordingly. Outcomes of these patients were assessed using mRS score.

Descriptive statistics were utilized and all results are presented in terms of percentages. Categorical data were compared using chi square test or fischer's exact test, if appropriate. Statistical significance was $p<0.05$.

SEX DISTRIBUTION

Sex	Frequency
Male	68
Female	32
Total	100

Table 2

A total of 100 patients were enrolled in the study, out of which 68 were male and 32 were female.

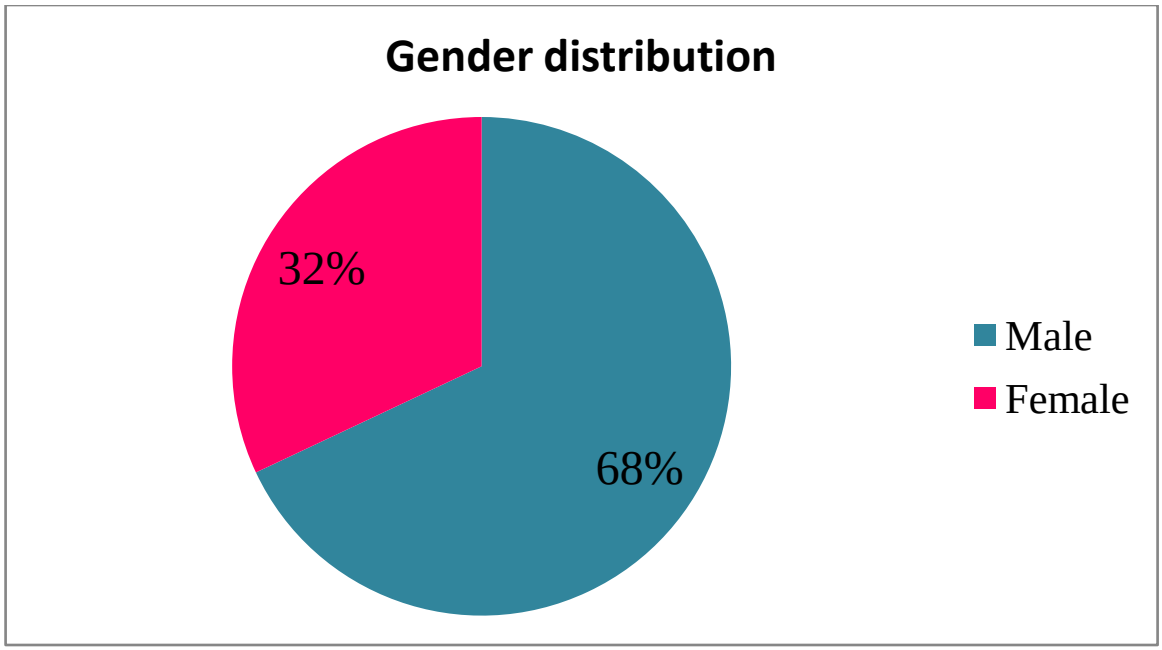

Figure 1

SEX DISTRIBUTION

Sex	Without Complications	With Complications
Male	36	32

Female	15	17
Total	51	49

Table 3

Among the 100 cases, 49 of them had at least one complication, out of which 32 were male and 17 were female. The rest had no complications (36 male and 15 female).

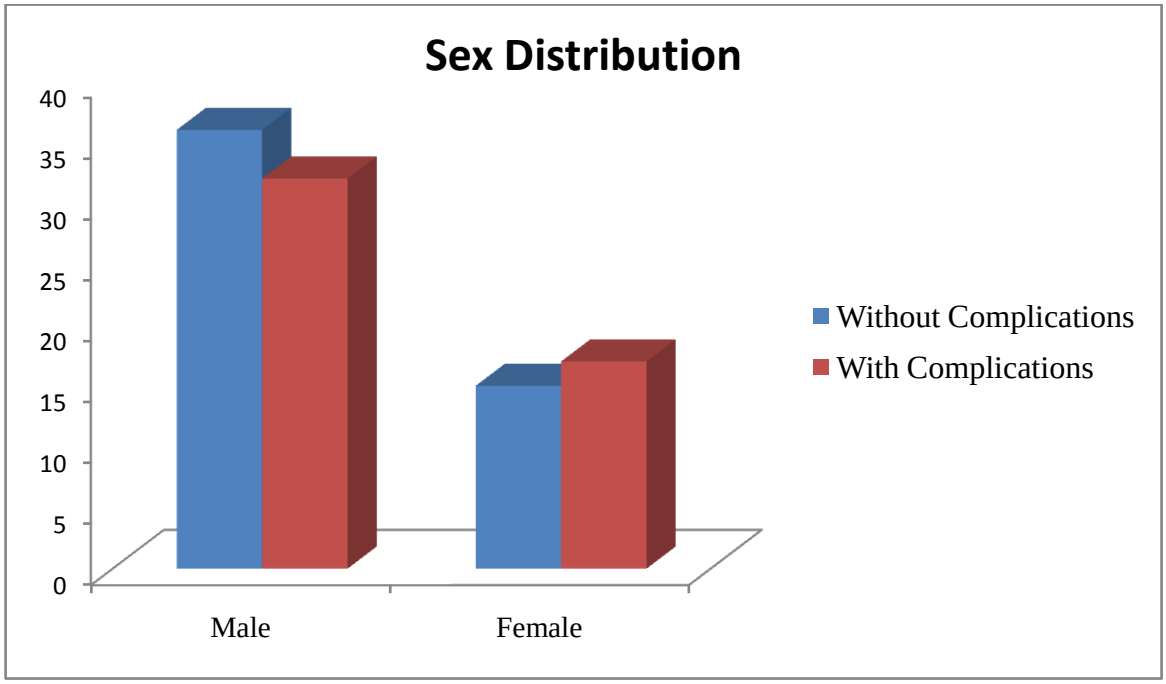

Figure 2

AGE DISTRIBUTION

Age	Without Complications	With Complications	Total
<50 yrs	20	24	44
>50 yrs	31	25	56

Table 4

The patients were divided into two age groups, less than 50 years and more than 50 years, with 44 and 56 patients in each group respectively. However there was no statistically significant association with regard to the age group and complications.

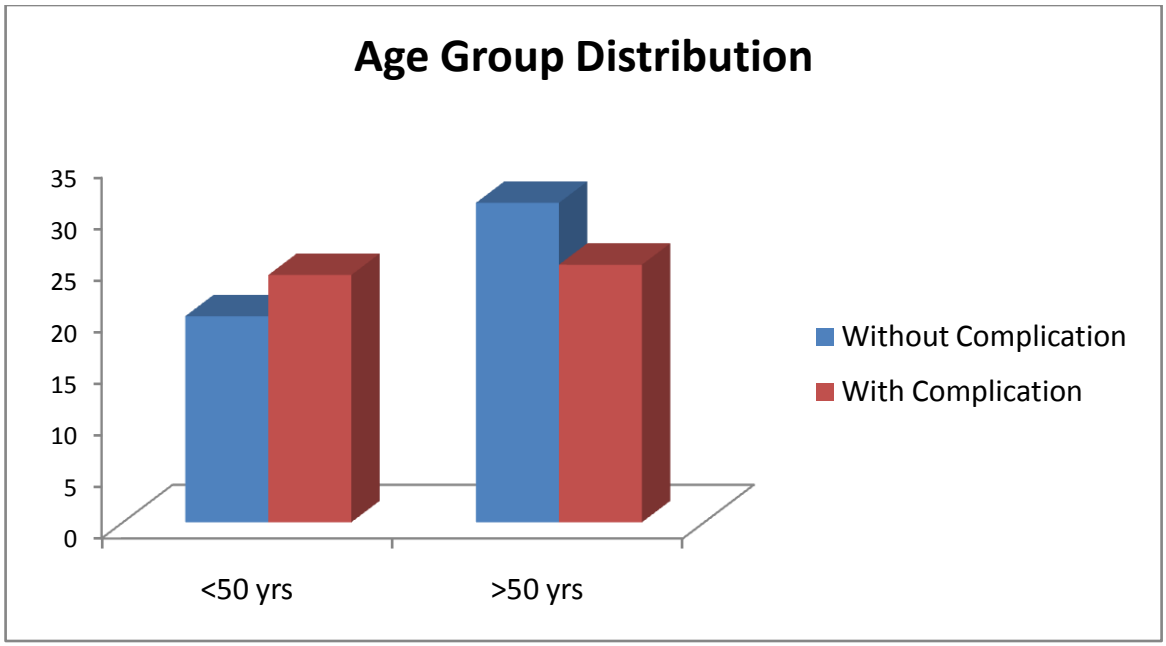

Figure 3

AGE DISTRIBUTION

Age in years	Male	Female	Total
20 – 30	5	2	7
31 – 40	11	2	13
41 – 50	14	9	23
51 – 60	24	6	30

61 – 70	11	10	21
71 - 85	3	3	6

Table 5

The age distribution of all the cases is shown in the above table. It ranges from 20 to 85 years. The mean age of the patients was 51.84±14 years. Distribution of the patients in the age group of 51-60 years was relatively higher than the other groups.

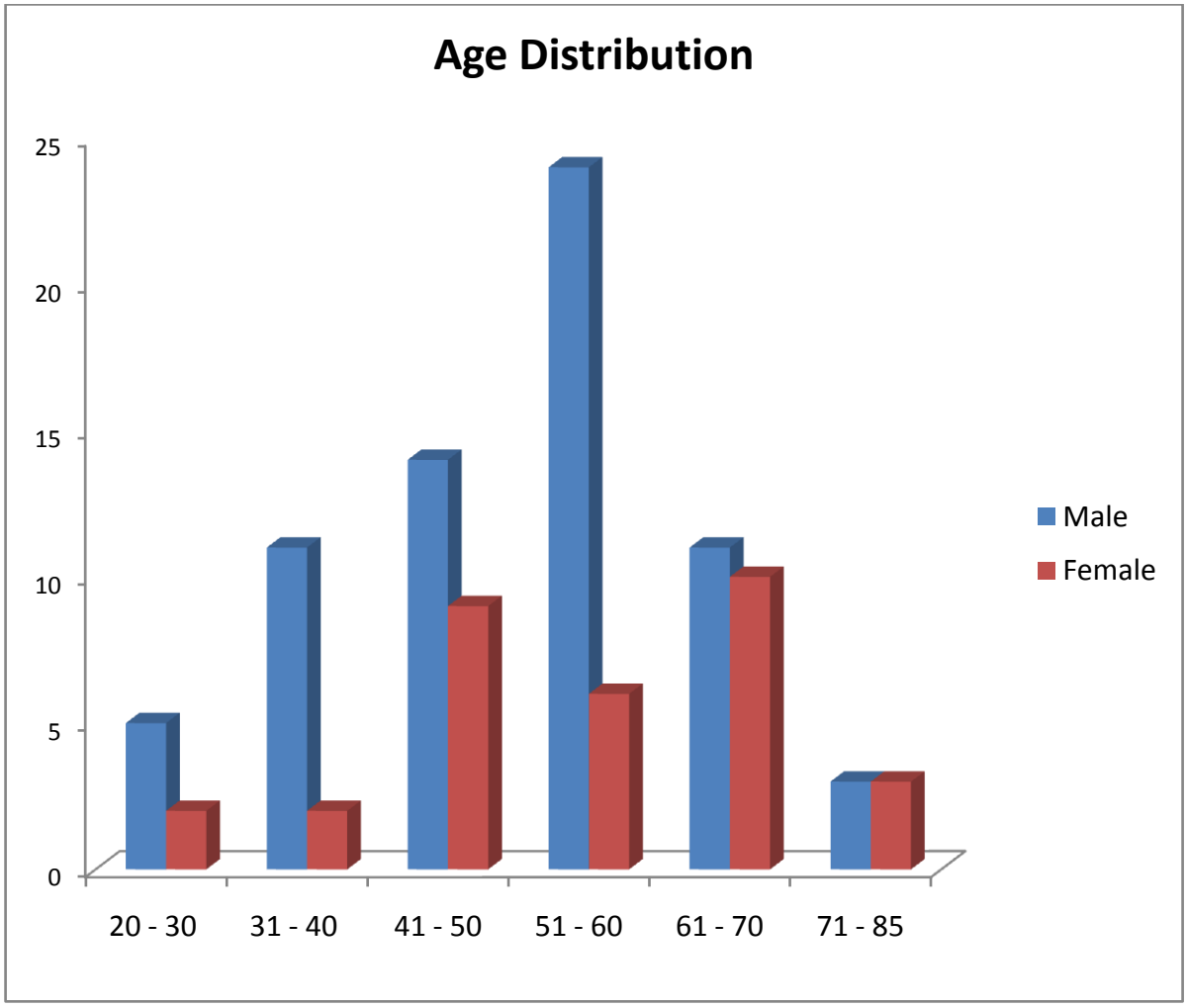
Age Distribution
25
20
15
10
5
0
20 - 30
31 - 40
41 - 50
51 - 60
61 - 70
71 - 85
Male
Female

Figure 4

STROKE SYMPTOMS ASSOCIATED WITH COMPLICATIONS

Symptoms	Without Complications	With Complications	P Value <0.05
Weakness			
Right	21 (41.2%)	22 (44.9%)	0.864
Left	22 (43.1%)	21 (42.9%)	
Dysphasia			
Absent	44 (86.3%)	34 (69.4%)	0.042
Present	7 (13.7%)	15 (30.6%)	
Dysarthria			
Absent	30 (58.8%)	19 (38.8%)	0.045
Present	21 (41.2%)	30 (61.2%)	

Dysphagia			
Absent	49 (96.1%)	35 (71.4%)	0.001
Present	2 (3.9%)	14 (28.6%)	
Loss of Sensation			
Absent	40 (78.4%)	43 (87.8%)	0.215
Present	11 (21.6%)	6 (12.2%)	
Unsteady gait			
Absent	45 (88.2%)	41 (83.7%)	0.511
Present	6 (11.8%)	8 (16.3%)	
Altered sensorium			
Absent	50 (98%)	46 (93.9%)	0.288
Present	1 (2%)	3 (6.1%)	
Vertigo			
Absent	46 (90.2%)	43 (87.8%)	0.697
Present	5 (9.8%)	6 (12.2%)	

Table 6

Various stroke symptoms were compared with complications and without complications. Statistically significant systems associated with complications were dysphagia (p-0.001), dysphasia (p-0.042), dysarthria (p-0.045), analyzed by chi square test.

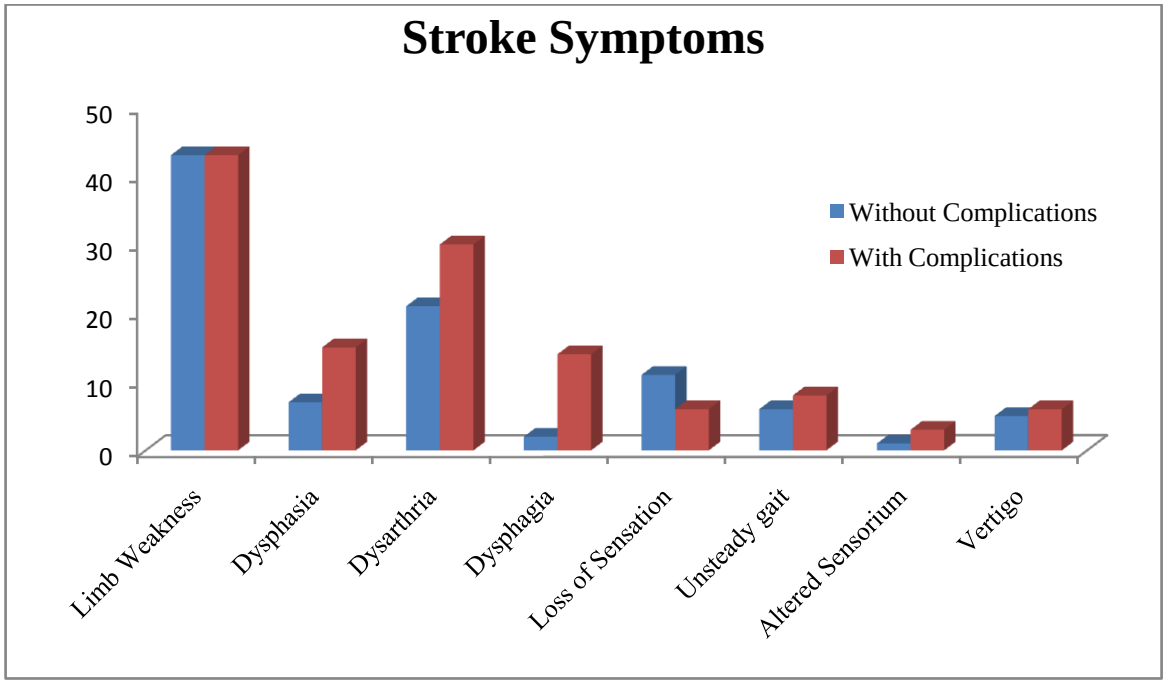

Figure 5

RISK FACTORS

Risk factors		Without Complications	With Complications	P value
HTN	Absent Present	28 (54.9%) 23 (45.1%)	27 (55.1%) 22 (44.9%)	0.984
DM	Absent Present	38 (74.5%) 13 (25.5%)	35 (71.4%) 14 (28.6%)	0.591
AF	Absent Present	49 (96.1%) 2 (3.9%)	49 (100%) 0	0.161
CAD	Absent Present	32 (62.7%) 19 (37.3%)	35 (71.4%) 14 (28.6%)	0.356
TIA	Absent Present	43 (84.3%) 8 (15.7%)	38 (77.6%) 11 (22.4%)	0.389
Smoking	Absent Present	27 (52.9%) 24 (47.1%)	23 (46.9%) 26 (53.1%)	0.548
Alcohol	Absent Present	32 (62.7%) 19 (37.3%)	27 (55.1%) 22 (44.9%)	0.437
Carotid Atherosclerosis	Absent Present	44 (86.3%) 7 (13.7%)	33 (67.3%) 16 (32.7%)	0.025

Table 7

Above table shows the various risk factors for acute ischemic stroke. Among them carotid artery atherosclerosis was statistically significant with complications after stroke. (p- 0.025)

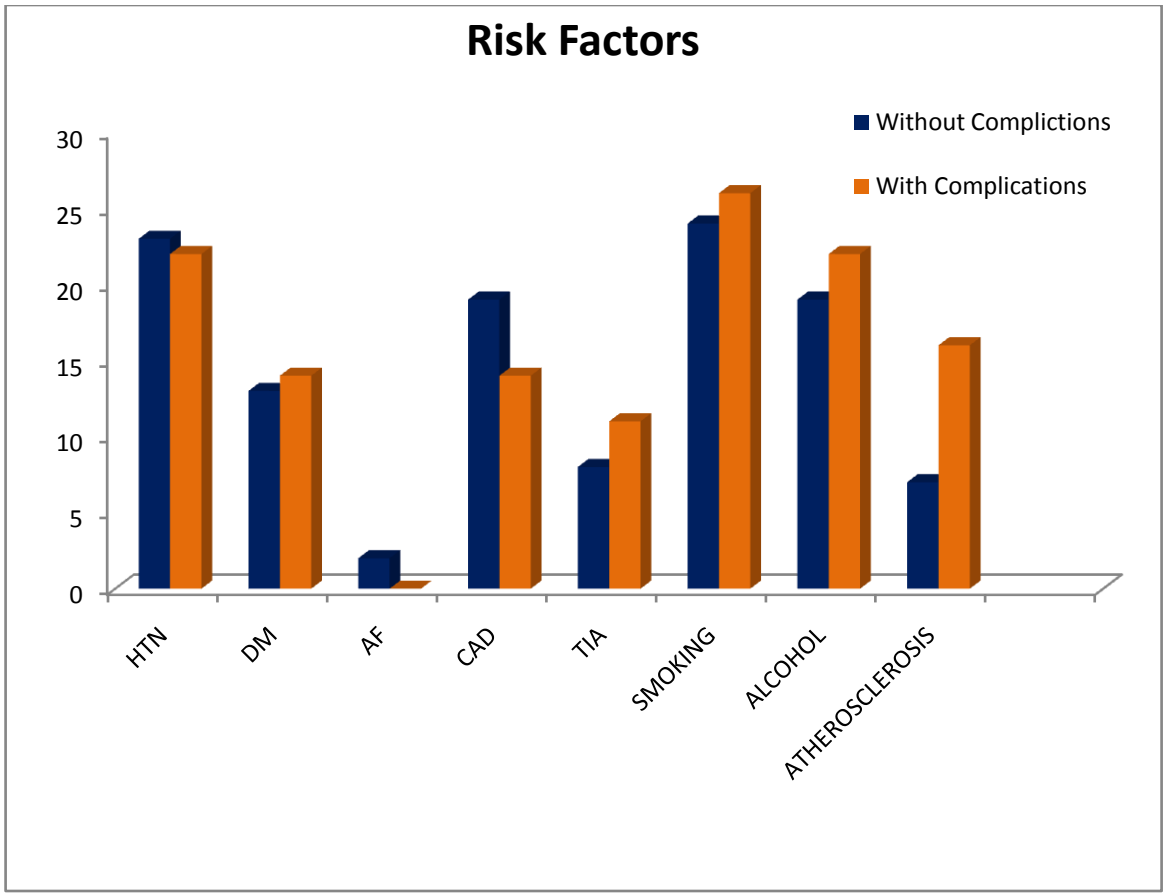
Risk Factors
Without Complictions
With Complications
30
25
20
15
10
5
0
HTN
DM
AF
CAD
TIA
SMOKING
ALCOHOL
ATHEROSCLEROSIS

Figure 6

LAB PARAMETERS

Lab Parameters		Without Complications	With Complications	P value
Hb	Normal Abnormal	18 (35.3%) 33 (64.7%)	25 (51%) 24 (49%)	0.112
RBS	<140 mg/dl >140 mg/dl	42 (82.4%) 9 (17.6%)	32 (65.3%) 17 (34.7%)	0.052
Urea	<45mg/dl >45mg/dl	50 (98%) 1 (2%)	44 (89.8%) 5 (10.2%)	0.083
Creatinine	<1.2 mg/dl >1.2 mg/dl	40 (78.4%) 11 (21.6%)	36 (73.5%) 13 (26.5%)	0.561
TGL	<150 >150	35 (68.6%) 16 (31.4%)	36 (73.5%) 13 (26.5%)	0.594
Total Protein	Normal Abnormal	42 (82.4%) 9 (17.6%)	42 (85.7%) 7 (14.3%)	0.647

Albumin	Normal Abnormal	23 (45.1%) 28 (54.9%)	22 (44.9%) 27 (55.1%)	0.984

Table 8

Various lab parameters were assessed in both the groups, with and without complications. Among them random blood sugar on admission was statistically significant in patients who had complications (p- 0.052).

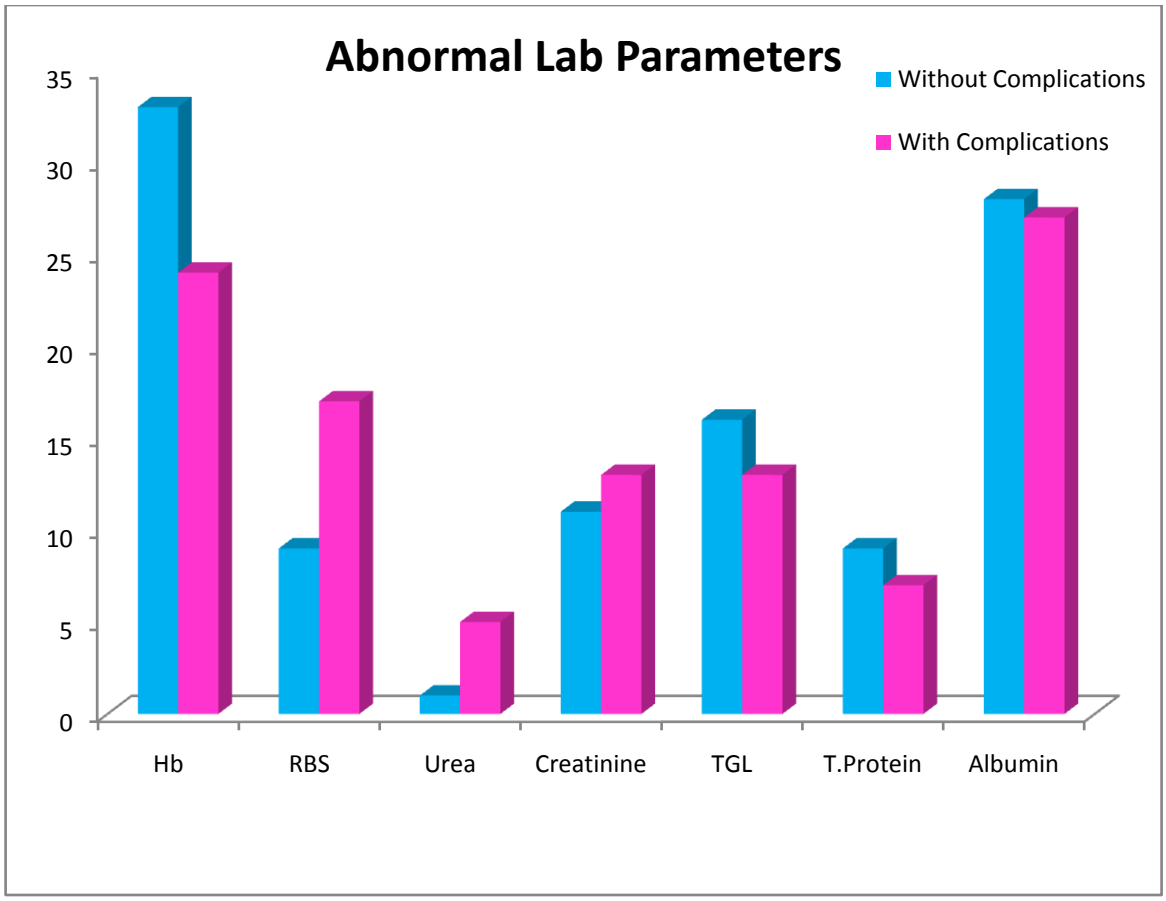
Abnormal Lab Parameters
Without Complications
With Complications
35
30
25
20
15
10
5
0
Hb
RBS
Urea
Creatinine
TGL
T.Protein
Albumin

Figure 7

STROKE CHARACTERISTICS

Variables		Without Complications	With Complications	P value
ACS		40 (78.4%)	40 (81.6%)	0.689
PCS		11 (21.6%)	9 (18.4%)	0.689
Hospital stays	≤10 days	25 (49%)	2 (4.1%)	0.000
	>10 days	26 (51%)	47 (95.9%)	
Stroke severity(NIHSS)	Mild-Moderate(0-10)	40 (78.4%)	16 (32.7%)	0.000
	Moderately severe-Severe(≥11)	11 (21.6%)	33 (67.3%)	
Functional outcome	Good(mRS-0-2)	23 (45.1%)	6 (4.2%)	0.000
	Poor(mRS>2)	28 (54.9%)	43 (87.8%)	

Table 9

Among the 100 patients 80% had anterior circulation stroke and the rest 20% were found to have posterior circulation stroke.

The average length of hospital stay was 11.7 ± 5.08 days for patients who had no complications and 19.88 ± 6.6 days for those who developed complications. 47 out of 49 (95.9%) patients who had complications were in the hospital for more than ten days, which is statistically significant (p- 0.000).

All the patients were grouped using NIHSS score into mild to moderate (0-10) and moderately severe to severe (>11). Among the patients who had complications, 33 were in the moderately severe to severe group which is statistically significant (p- 0.000).

Functional outcome of these patients were assessed at the end of one month after stroke and grouped as good (≤2) and poor (>2) outcome according to mRS score. Out of 49 patients who had complications 43 (87.8%) had poor outcome which is statistically significant (p- 0.000) when compared to those who had no complications (54.9%).

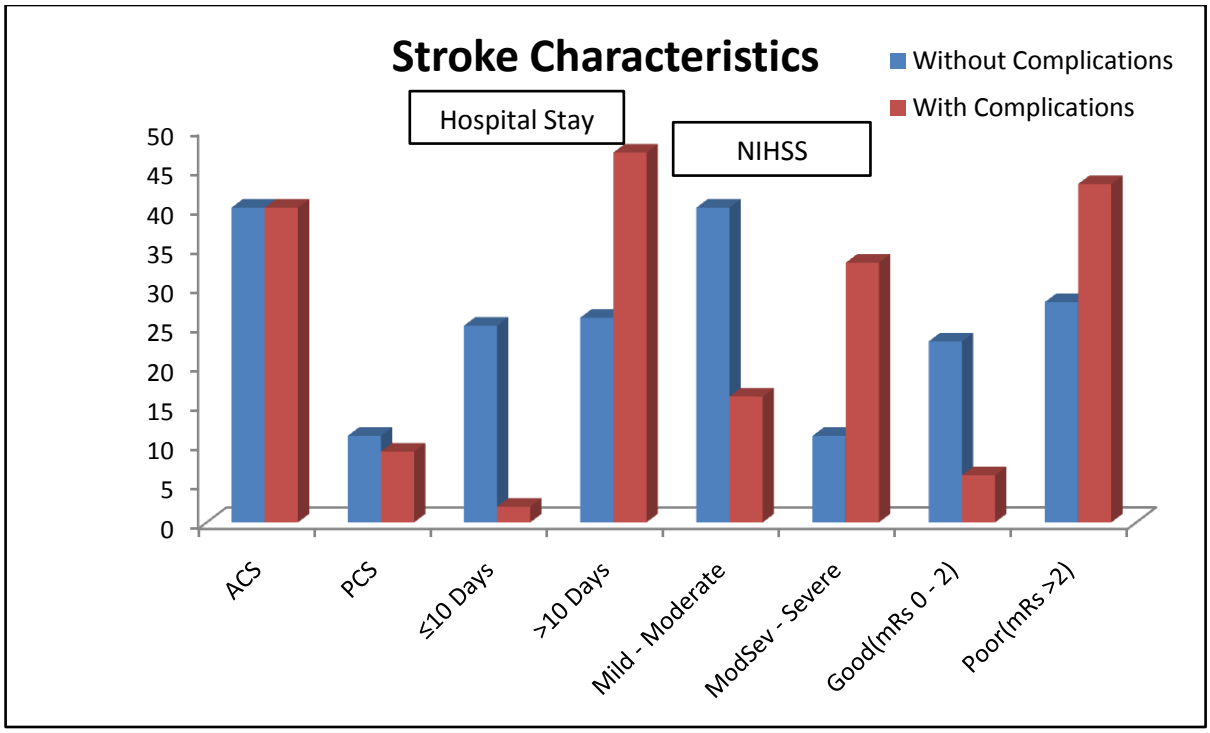

Figure 8

COMPLICATIONS IN ACUTE ISCHEMIC STROKE

Complications	Percentage
Respiratory tract infection	20
Urinary tract infection	24
Bed sore	23
Dysphagia	28
Constipation	40
UGI bleed	1
Seizure	1
Haemorrhagic transformation with oedema	10
Depression	22
Shoulder pain	31
Hematuria	4
Hyponatremia	1

Patients with at least one complication	49

Table 10

Complications noted during the hospital stay are listed in the above table. Out of the 100 patients, 49 had at least one complication. The most commonly found complications in their order of frequency were constipation, shoulder pain, dysphagia, UTI, bed sore, depression and RTI. Neurological complications in the form of haemorrhagic transformation with brain oedema were found in 10 patients and seizure in one patient.

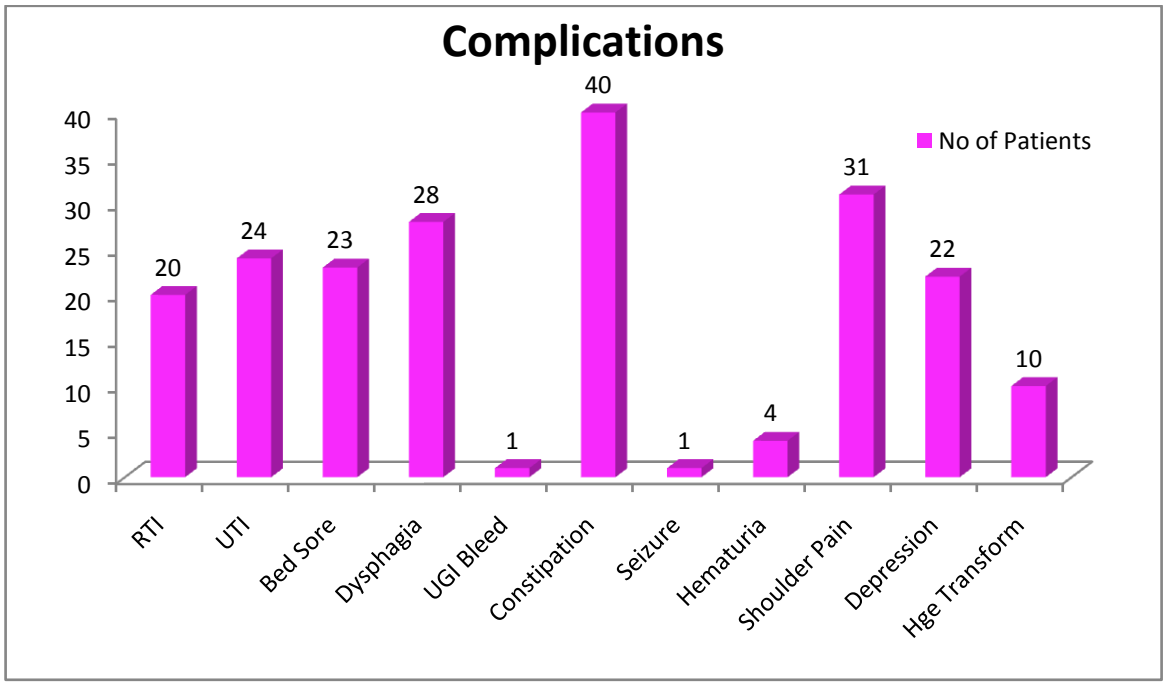

Figure 9

DISCUSSION

The purpose of our study was to describe the occurrence of various complications in acute ischemic stroke patients and also assess the outcome of the patients using mRS score at admission, discharge and at the end of one month after stroke.

In our study 49% of the patients developed at least one complication during their hospital stay; however the frequency of complications reported in previous studies varies from 40% to 95%.[6,7,8,9] This wide variation may be due to the following reasons- no standard definitions for complications following the stroke, varied follow-up periods ranging from 1 week to 30 months, type and severity of stroke affecting the incidence and finally, some of the variations may be due to the demographic variables.

In our study the occurrence of complications in their order of frequency was constipation (41%), shoulder pain (31%), dysphagia (28%), UTI (24%), bed sore (23%), depression (22%), RTI (20%), haemorrhagic transformation with brain oedema (10%), hematuria (4%), UGI bleed (1%), seizure (1%) and hyponatremia (1%), with no incidence of thromboembolic complications. Only one patient was deceased at the end of one month follow-up.

Constipation

In our study, 41% of the patients had constipation following an ischemic stroke which is unlike the study of Jose C Navarro et al who reported constipation in only 7.9% of his patients.[77]

Shoulder pain

31% of the patients had shoulder pain in our study which is similar to the study by Braus DF et al [72] who found shoulder pain in 27% of his patients. Lindgren I et al [71] and Jeyaraj D Pandian et al[10] showed the occurrence of shoulder pain to be around 22% and 7.8% respectively in their study.

Dysphagia

Mann G et al [78] and Ramsey DJ et al[61] had dysphagia in 51% and 50% of patients respectively whereas it was only 28% in our study. The incidence was

increased to 64% in the study by Mann G et al when video fluoroscopy was used for the diagnosis of dysphagia.

Infection

Commonly found infections were that of the urinary tract (24%) and respiratory tract (20%) which is similar to Katarzyna Grabska et al [79] with 19.5% of RTI and 23.3% of UTI and Jeyaraj D Pandian et al with 21.2% of RTI. But the incidence of UTI was only 8.7% in their study. In contrast to our study, Jose C Navarro et al had a lesser incidence with 9.4% of RTI and 4.9% of UTI.

Bed sore

23% of patients in our study developed bed sores which are comparatively more to that of Jeyaraj D pandian et al and Jose C Navarro et al with only 7.8% and 2.6% respectively.

UGI bleed

Frequency of UGI bleed was 1% in our study which is comparable to that of Jose C Navarro with 1.6%. Study of Jeyaraj D Pandian showed 2.23% and that of Davenport RJ [69] showed 3% of UGI bleed.

Seizure

1% of patients in our study reported to have seizures after stroke which is similar to that of Bogousslavsky J et al[80] with less than 1% and Jose C Navarro et al with 1.3%. However Jeyaraj D Pandian et al found a higher incidence about 3.8% in their study.

Hematuria

Frequency of hematuria was found to be higher in our study (4%) when compared to 1.1% in the study of Jose C Navarro.

Depression

Studies with similar frequency with that of ours (22%) were Astrom M et al [81] (25%) and Jeyaraj D Pandian et al (18.1%). A lesser frequency (4%) was found in the study of Jose C Navarro et al.

Haemorrhagic transformation with brain edema

Incidence of hemorrhagic transformation was 10% in our study which is similar to that found in Krieger DW et al[25] (10%-20%) but it was much higher (30%- 40%) in Lyden PD et al.[82]

Hyponatremia

Study of Luisa Fofi et al[83] showed an incidence of hyponatremia in 6.3% of survivors and 20.7% of the deceased following an acute ischemic stroke. Similar findings of about 4% were found in our study. Study of Sreeraj K et al showed hyponatremia in 31.1% of their patients[84]. Sajadieh A et al demonstrated that a poor outcome was three times more likely in patients with hyponatremia.[85]

Lab parameters

Though the prevalence of anaemia (57%) was more in the study group, it did not influence the complications. Abnormal sugar values (more than 140 mg/dl) was found to influence the complication with a frequency of 26%. This is similar to the study of Wong AA et al where the frequency of abnormal blood sugar (more than 126 mg/dl) was up to 50%.[86]

Other parameters like serum creatinine, triglycerides, total proteins and albumin did not have any influence over the complications.

Strength of our present study is that it is a prospective study design which can provide more accurate information than a retrospective study. This is one among the few studies with data on majority of complications confined to ischemic

stroke from a particular group of population. However there are few limitations in our study like non assessment of severity of complications, time of occurrence of stroke.

SUMMARY

The results of the present study showed that the incidence of complications in patients after an acute ischemic stroke to be 49%.

The length of hospital stay, blood sugar, carotid artery atherosclerosis, severity of stroke (NIHSS score), dysphagia and dysarthria were the factors with significant association with complications.

The complications encountered were constipation (40%), shoulder pain (31%), dysphagia(28%), UTI (24%), bed sore (23%), depression (22%), respiratory tract infection (20%), haemorrhagic transformation with brain oedema (10%), hematuria (4%), hyponatremia (1%), UGI bleed(1%) and seizures (1%).

Patients with complications were more likely to have poor outcome (87.8%) compared to patients without complications (54.9%).

Only 1% of patients were found to be deceased during follow up at the end of one month.

CONCLUSION

In our study, the rate of medical complications was high in patients with acute ischemic stroke and those patients were found to have a poor outcome. Hence early diagnosis and prompt treatment of these complications is needed, in order to improve the outcome and thereby the quality of life of patients after stroke.

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ABBREVIATIONS

HTN	Hypertension
DM	Diabetes Mellitus
AF	Atrial Fibrillation
CAD	Coronary Artery Disease
TIA	Transient Ischemic Attacks
Hb	Haemoglobin
RBS	Random Blood Sugar
TGL	Triglyceride
TCH	Total Cholesterol
ACS	Anterior Circulation Stroke
PCS	Posterior Circulation Stroke
UGI Bleed	Upper Gastro Intestinal Bleed
RTI	Respiratory Tract Infection
UTI	Urinary Tract Infection
DVT	Deep Vein Thrombosis
PE	Pulmonary Embolism
LMWH	Low Molecular Weight Heparin
VTE	Venous Thrombo Embolism
HgeTr	Haemorrhagic Transformation

PROFORMA

NAME AGE SEX

CLINIC NO: ADM NO: WARD

DOA DOD

ADDRESS:

PHONE NO:1. 2.

LITERACY:

OCCUPATION : INCOME:

SOCIO ECONOMIC STATUS:

Admission from 1.community 2.other hospital 3.emergency ward

CLINICAL HISTORY:

RISK FACTORS

H/O Hypertension - Yrs on admission BP

H/O Diabetes mellitus- yrs

H/O valvular heart disease prosthetic/ rheumatic

H/O arrhythmia- AF

H/O MI /IHD

Heart failure

Past H/O stroke Family history H/O stroke

TIA PAD

Tobacco cigarette beedi yrs

Alcohol ocp drug abuse

GENERAL PHYSICAL EXAMINATION

PR:

BP: SBP DBP

RR: TEMP:

SYSTEMIC EXAMINATION:

CVS:

RS:

ABDOMINAL EXAMINATION:

CNS:

NIHSS Score:

Modified Rankin Scale Score: Admission: At Discharge: 1month

POST STROKE COMPLICATION

1.INFECTION- symptoms

Respiratory

Urinary tract

Bedsore site grade

2.GIT COMPLICATION

Dysphagia

UGI BLEED

3.DVT RT/LT

4.DEPRESSION

5.NEUROLOGIC – Recurrent stroke/Seizure

6.OTHERS

INVESTIGATION

Hb			
TLC			
DC-P			
L			
M			
E			
ESR			
PLAT			
PCV			
FBS			
PPBS			
RBS			
UREA			
CREAT			

Na			
K			
LDL			
T.CH			
TGL			
HDL			
BILIRU			
OT			
PT			
SLP			
T.PROT			
A/G			

URINE R/E

URINE C/S

SPUTUM C/S

PUS C/S

SWAB C/S

CXR

ECG

ECHO

CT BRAIN MCA LT RT
PCA
ACA

MRI BRAIN

Carotid & vertebral Doppler

TEMP CHART D1 D2 D3 D4 D5 D6 D7 D8

ANTIBIOTICS

PATIENT CONSENT FORM

Study Details : **Complications and outcome of acute ischaemic stroke.**

Study Centre : **Rajiv Gandhi Government General Hospital, Madras Medical College, Chennai - 600 003.**

Patient may check (✓) these boxes:

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ☐

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ☐

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulartory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ☐

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms. ☐

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, microbiological, radiological tests, carotid and vertebral doppler. ☐

I hereby consent to participate in this study. ☐

Signature / Thumb impression :

Patient Name and Address : Place : Date :

Signature of Investigator

Study Investigator's Name : Place : Date :

சுய ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு

ளைக்கு செல்லும் இரத்த குழாய் அடைப்பினால் ஏற்படும் பக்கவாதத்தின் பாதிப்புகள் மற்றும் விளைவுகள் பற்றிய ஆய்வு.

ஆராய்ச்சி நிலையம் : இராஜீவ் காந்தி அரசு பொது மருத்துவமனை மற்றும் சென்னை மருத்துவக் கல்லூரி, சென்னை - 600 003.

பங்கு பெறுபவரின் பெயர் : உறவுமுறை :

பங்கு பெறுபவரின் எண். :

பங்கு பெறுபவர் இதனை (✓) குறிக்கவும்

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களைப் பெறவும் வாய்ப்பளிக்கப்பட்டது. ☐

நான் இவ்வாய்வில் தன்னிச்சையாகத்தான் பங்கேற்கிறேன். எந்தக் காரணத்தினாலோ எந்தக் கட்டத்திலும் எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகிக் கொள்ளலாம் என்றும் அறிந்து கொண்டேன். ☐

இந்த ஆய்வு சம்மந்தமாகவோ, இதை சார்ந்த மேலும் ஆய்வு மேற்கொள்ளும்போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளைப் பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன். ☐

இந்த ஆய்வின் லம் கிடைக்கும் தகவல்களையும், பரிசோதனை முடிவுகளையும் மற்றும் சிகிச்சை தொடர்பான தகவல்களையும் மருத்துவர் மேற்கொள்ளும் ஆய்வில் பயன்படுத்திக் கொள்ளவும் அதைப் பிரசுரிக்கவும் என் முழு மனதுடன் சம்மதிக்கிறேன். ☐

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். எனக்குக் கொடுக்கப்பட்ட அறிவுரைகளின்படி நடந்து கொள்வதுடன், இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ அல்லாத எதிர்பாராத வழக்கத்திற்கு நோய்க்குறி தென்பட்டாலோ உடனே அதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்கிறேன். ☐

இந்த ஆய்வில் எனக்கு இரத்தம், சிறுநீர், சளி, **Chest X-Ray, CT Scan/ MRI Scan/ Doppler** பரிசோதனை செய்து கொள்ள நான் முழு மனதுடன் சம்மதிக்கிறேன். ☐

பங்கேற்பவரின் கையொப்பம்..... இடம்..... தேதி
கட்டைவிரல் ரேகை

பங்கேற்பவரின் பெயர் மற்றும் விலாசம்.....

ஆய்வாளரின் கையொப்பம்..... இடம்..... தேதி

ஆய்வாளரின் பெயர்.....

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No: 04425305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To

Dr. C. Chennappan
PG in DM Neurology
Madras Medical College, Chennai -3

Dear Dr. C. Chennappan

The Institutional Ethics Committee of Madras Medical College reviewed and discussed your application for approval of the proposal entitled "Complications and outcome of acute ischemic stroke" No. 32052012.

The following members of Ethics Committee were present in the meeting held on 30.05.2012 conducted at Madras Medical College, Chennai -3.

1.	Prof. S.K. Rajan, MD	-- Chairperson
2.	Prof. Pregna B. Dolia MD Vice Principal , Madras Medical College, Chennai -3 Director , Instt.of Bio Chemistry, MMC, Ch-3	-- Member Secretary
3.	Prof R. Nandhini, MD Director, Institute of Pharmacology, MMC, Ch-3	-- Member
4.	Prof. P. Karkuzhali MD Director i/c Prof & Head , Dept. of Pathology, MMC, Ch-3	-- Member
5.	Prof.A. Radhakrishnan MD Prof. of Internal Medicine, MMC, Ch-3	-- Member
6.	Prof. P. Raghumani MS Prof. of Surgery, Dept. of Surgery, MMC, Chennai -3	-- Member
7.	Thiru. S. Govindasamy . BA.BL	-- Lawyer
8.	Tmt. Arnold Soulina MA	-- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd /. Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information / informed consent and asks to be provided a copy of the final report

Member Secretary, Ethics Committee

KEY TO MASTER CHART

0	- Absent
1	- Present
ACS 1	- Anterior Circulation Stroke
PCS 1	- Posterior Circulation Stroke

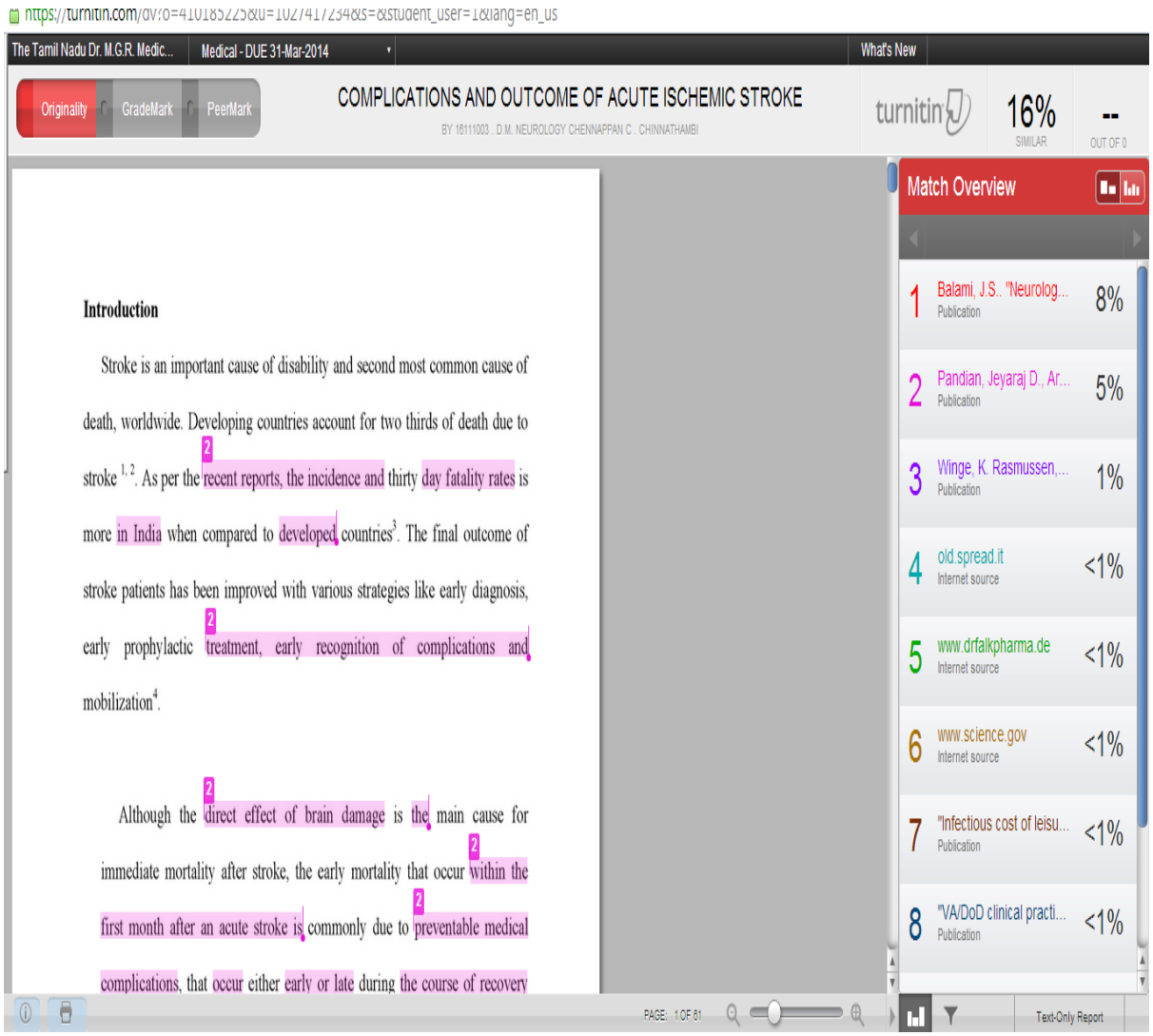
OUTCOME

0	- Good Outcome
1	- Poor Outcome

SNO	NAME	AGE	SEX	NO. OF HOSP STAY	WEAKNESS	LOSS OF SENSATION	DYSPHASIA	DYSPHAGIA	DYSARTHR	UNSTEADY	ALTSENSOR	VERTIGO	HTN	DM	AF	CAD	TIA	SMOKING	ALCOHOL	DOPPLER	MRS ADMISSION	DISCHARGE	1MONTH	NIHSS	RTI	UTI	BED SORE	DYSPHAGIA	UGI BLEED	SEIZURE	CONSTIPATION	HEMATURIA	SHOULDER PAIN	DEPRESSION	HgeTr	Hb	RBS	UREA	CREATININ	TCh	TGL	T.PROT	ALB	PCS	ACS	Outcome
1	Rajendran	37	m	19	1	0	1	0	0	0	0	0	0	0	0	0	0	1	1	0	5	4	4	16	0	0	0	0	0	0	1	1	1	1	0	15.8	89	24	0.7	168	85	6.8	3.6	0	1	1
2	Devaraj	50	m	21	1	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	5	5	5	16	1	1	1	1	0	0	1	0	1	1	0	10.9	87	38	1.2	172	110	6.6	3.7	0	1	1
3	Valli	65	f	13	1	0	1	0	0	0	0	0	1	1	0	1	0	0	0	0	5	5	5	14	1	1	1	1	0	0	1	0	0	0	1	12.9	142	24	0.7	229	463	6.8	3.9	0	1	1
4	Ponnusamy	24	m	15	1	0	0	0	0	0	0	0	1	0	0	0	0	1	1	0	4	3	2	11	0	0	0	0	0	0	1	0	1	1	1	17.3	237	26	0.8	160	112	6.9	3.8	0	1	0
5	Udayakumar	35	m	16	2	0	0	0	0	0	0	0	0	0	0	0	1	1	1	1	5	4	4	13	0	0	1	0	0	1	1	0	1	1	0	13.7	112	34	1	145	89	6.7	3.6	0	1	1
6	Sekar	38	m	31	2	0	0	0	1	0	0	0	0	0	0	0	0	1	1	0	5	4	4	15	0	1	1	1	0	0	1	0	1	1	0	9.8	96	26	0.8	159	122	6.4	3.5	0	1	1
7	Meerabai	65	f	18	1	0	0	0	1	0	0	0	0	1	0	0	1	1	0	1	4	3	2	7	0	0	0	1	0	0	1	0	1	0	0	12.3	149	26	0.7	165	160	6.1	3.1	1	0	0
8	Joseph	40	m	19	2	0	0	0	1	1	1	0	1	1	0	0	0	1	1	0	3	2	2	6	0	0	0	0	1	0	0	0	0	0	0	16.5	215	18	0.9	135	142	6.7	3.5	1	0	0
9	Rajeswari	52	f	22	2	0	0	1	1	0	0	0	1	1	0	1	1	0	0	0	5	5	4	13	1	1	1	1	0	0	1	0	1	1	0	13.3	259	38	1	152	99	6.9	4.2	0	1	1
10	Kamala	65	f	24	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	1	4	4	2	11	0	1	0	0	0	0	1	0	0	1	0	13.6	78	26	0.9	163	110	6.3	3.5	1	0	0
11	Varadharajalu	57	m	31	0	1	0	1	1	1	0	1	0	0	0	0	0	1	1	1	4	3	2	4	1	0	0	1	0	0	1	0	0	1	0	10.4	80	20	1	168	97	6.2	3.4	1	0	0
12	Shantha	70	f	19	1	0	1	0	1	0	0	0	1	1	0	1	1	0	0	1	5	4	4	10	0	1	1	0	0	0	1	0	0	1	0	12.6	135	28	1.1	188	113	6.4	3.3	0	1	1
13	Ramesh	34	m	23	2	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	5	5	4	13	0	0	1	1	0	0	1	0	0	1	0	14.2	98	23	0.8	169	114	6.9	3.7	0	1	1
14	Subramani	70	m	25	2	0	0	0	1	0	0	0	1	0	0	1	1	1	0	1	5	5	5	16	0	0	1	1	0	0	1	0	0	1	0	13.9	83	28	1	175	110	6.8	3.9	0	1	1
15	Nagarajan	48	m	21	2	0	0	0	1	0	0	0	0	0	0	0	0	1	1	0	4	4	4	11	0	0	0	0	0	0	0	0	1	0	1	15.6	200	26	1.2	106	238	6.4	3.4	0	1	1
16	Kannaiyan	52	m	24	1	0	0	0	1	0	0	0	1	1	0	1	1	1	1	0	4	3	3	9	1	0	0	0	0	0	1	0	1	0	0	10.6	186	22	0.7	152	164	7	3.7	0	1	1
17	Kothandan	48	m	22	0	1	0	1	1	1	0	1	0	0	0	0	0	1	0	0	4	4	4	6	1	0	0	1	0	0	0	0	0	0	0	15	114	36	1.2	105	116	6.8	3.2	1	0	1
18	Sundaramoorthy	63	m	14	1	0	1	0	1	0	0	0	0	0	0	0	0	1	1	1	5	5	4	17	0	1	0	1	0	0	1	0	1	0	0	14.1	138	34	1.2	104	55	6.4	3.2	0	1	1
19	Pandiyan	55	m	41	1	0	1	1	1	0	1	0	0	0	0	0	0	1	1	0	5	5	5	16	1	1	1	1	0	0	1	0	1	1	0	12.1	86	32	1.1	185	176	6.4	2.8	0	1	1
20	Sethupathi	56	m	16	2	0	0	0	1	0	0	0	0	1	0	0	1	1	0	1	5	5	4	16	1	0	0	1	0	0	1	0	1	0	0	13.5	160	24	0.8	185	166	6.5	3.2	0	1	1
21	Sangeet	35	m	21	1	0	1	0	1	0	0	0	0	0	0	1	0	1	1	0	5	4	4	17	1	0	0	1	0	0	1	0	1	0	1	13.3	85	46	1.5	140	121	6.6	3.7	0	1	1
22	Palaniammal	75	f	8	2	0	0	0	0	0	0	0	1	1	0	0	1	0	0	0	4	4	4	10	0	0	0	1	0	0	1	0	1	0	0	8.3	209	30	1.2	143	128	6.2	3	0	1	1
23	Poothanam	65	f	15	2	0	1	0	1	0	0	0	1	0	0	1	0	0	0	1	5	4	4	17	1	0	1	1	0	0	1	0	1	0	1	10.8	84	18	0.5	268	175	5.6	3.1	0	1	1
24	Ellamalli	70	f	9	2	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	4	4	3	10	1	0	1	0	0	0	1	0	1	0	0	4.3	108	23	0.7	197	145	7.7	3	0	1	1
25	Logeshwari	21	f	14	1	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	5	4	2	17	0	0	1	1	0	0	1	0	1	0	0	8.8	146	25	0.9	122	104	6	3	0	1	0
26	Rambai	50	f	15	1	0	1	0	0	0	0	0	0	1	0	1	0	0	0	1	4	4	3	15	0	1	1	1	0	0	1	1	0	0	0	13.7	106	26	0.9	185	145	6.8	3.7	0	1	1
27	Rajendran	37	m	19	1	0	1	0	0	0	0	0	0	0	0	0	0	1	1	0	5	4	4	16	0	0	0	0	0	0	1	1	1	1	0	15.8	89	24	0.7	168	85	6.8	3.6	0	1	1
28	Stalin	45	m	12	2	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	5	5	4	15	0	1	1	1	0	0	1	0	1	0	1	12.6	80	34	1.2	189	157	7	3.8	0	1	1
29	Velankanni	45	f	17	2	0	0	1	1	0	1	0	0	0	0	0	0	1	1	0	5	4	4	14	0	0	1	1	0	0	1	0	1	0	1	7.8	122	23	0.8	248	251	5.8	2.5	0	1	1
30	Muniyammal	48	f	18	2	0	0	0	0	1	0	0	1	0	0	0	0	0	0	0	4	4	3	14	0	0	1	0	0	0	1	0	1	0	0	10.1	95	22	0.9	134	125	5.3	3.2	0	1	1
31	Ravi	40	m	20	0	1	0	1	1	1	0	1	1	0	0	0	0	0	0	0	4	4	4	5	1	1	0	0	0	0	1	0	0	1	0	16	99	30	1	134	120	5.9	3	1	0	1
32	David Rajendran	56	m	21	2	1	0	1	1	0	0	0	1	0	0	0	0	0	1	0	5	4	4	11	1	1	1	0	0	0	1	0	1	0	0	12.8	149	48	1.3	163	147	5.8	3.1	0	1	1
33	Kannayan	60	m	23	1	0	1	1	0	0	0	0	0	0	0	1	1	0	0	0	5	4	4	12	0	1	1	0	0	0	1	0	1	0	0	11.9	84	39	1.3	184	99	6.4	3.4	0	1	1
34	Jaganathan	70	m	30	0	0	0	1	1	1	0	1	1	1	0	0	0	0	0	0	4	4	4	5	1	1	0	1	0	0	0	0	0	1	0	11	170	58	1.1	198	145	6.5	3.3	1	0	1
35	Krishnaveni	59	f	14	1	0	1	0	0	0	0	0	1	1	0	0	0	0	0	0	5	4	4	11	1	1	0	0	0	0	1	0	1	1	0	11.3	241	83	3	198	142	5.3	2.9	0	1	1
36	Gunapoosam	63	f	14	2	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	5	4	4	13	1	1	0	0	0	0	1	0	1	1	0	10.8	107	48	2.8	174	164	6.7	3	0	0	1
37	Venkateshan	55	m	10	1	0	1	0	0	0	0	0	1	0	0	0	0	1	1	0	5	4	4	15	0	1	0	0	0	0	1	0	1	1	0	13.2	110	35	1.6	195	122	6	3.2	0	1	1
38	Nagavel	65	m	16	2	0	0	0	0	0	0	0	1	0	0	1	0	0	1	1	4	4	3	9	0	1	1	1	0	0	1	0	1	0	0	13.7	52	24	1.2	149	110	6.9	3.4	0	1	1
39	Sekar	38	m	31	2	0	0	0	1	0	0	0	0	0	0	0	0	1	1	0	5	4	4	15	0	1	1	1	0	0	1	0	1	1	0	9.8	96	26	0.8	159	122	6.4	3.5	0	1	1

40	Ezhumalai	30	m	15	1	0	0	1	1	0	0	0	1	0	0	0	0	0	0	0	5	5	4	11	1	1	0	1	0	0	0	0	0	1	0	12.5	180	21	0.7	168	118	6.6	3	0	1	1
41	Kothandan	48	m	22	0	1	0	1	1	1	0	1	0	0	0	0	1	1	0	0	4	4	4	6	1	0	0	1	0	0	0	0	0	0	0	15	114	36	1.2	105	116	6.8	3.2	1	0	1
42	Kannan	48	m	35	0	1	0	1	1	1	0	1	0	0	0	0	0	1	1	0	4	4	4	6	1	0	0	1	0	0	1	0	0	0	0	15.2	165	35	1.2	113	198	6.8	3.4	1	0	1
43	Sabari nisha	42	f	24	1	0	1	1	0	0	0	0	1	1	0	0	0	0	0	1	4	3	3	10	1	1	1	0	0	0	1	0	1	1	0	11.3	130	24	0.8	140	112	6.7	3.9	0	1	1
44	Mary	50	f	13	1	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	4	3	3	6	0	0	0	0	0	0	0	0	0	0	1	10.5	87	39	1	158	128	6.6	3.6	0	1	1
45	Surya moorthy	60	m	20	1	0	1	0	1	0	0	0	1	1	0	1	1	1	1	0	5	4	4	11	0	0	0	0	0	0	0	1	0	0	0	10.4	246	28	0.9	163	108	6.9	3.5	0	1	1
46	Subramani	70	m	25	2	0	0	0	1	0	0	0	1	0	0	1	0	1	0	1	5	5	5	16	0	0	1	1	0	0	1	0	0	1	0	13.9	83	28	1	175	110	6.8	3.9	0	1	1
47	Munusamy	54	m	16	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	1	5	4	4	15	0	1	1	1	0	0	1	0	1	0	0	10.9	102	20	0.7	390	198	6.9	4.2	0	1	1
48	Kesavan	85	m	20	2	0	0	1	1	0	0	0	1	0	0	1	0	0	0	1	5	4	4	11	0	1	0	1	0	0	1	0	1	0	1	14.8	145	35	1.2	192	145	6.7	3.2	0	1	1
49	Vijaya lakshmi	48	f	23	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4	4	4	9	0	1	0	0	0	0	0	0	0	0	1	9.7	102	18	0.9	208	167	5.3	3.3	0	1	1
50	Jeya	35	f	17	2	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	5	3	2	13	0	0	0	0	0	0	0	0	0	0	0	10.5	110	32	1	150	131	6.6	3.7	0	1	0
51	Nannappan	76	m	5	1	0	1	0	1	0	0	0	1	1	0	1	0	0	1	0	4	4	3	10	0	0	0	0	0	0	0	0	0	0	0	12.8	131	21	0.8	180	110	6.9	3.2	0	1	1
52	Lucas	60	m	8	2	0	0	0	0	0	0	0	1	0	0	0	0	1	1	0	2	2	1	2	0	0	0	0	0	0	0	0	0	0	0	13.8	82	26	1	160	112	6.6	3.7	0	1	0
53	Kandhan	55	m	5	1	0	1	0	0	0	0	0	0	0	0	0	1	1	1	0	3	3	2	7	0	0	0	0	0	0	0	0	0	0	0	11.4	95	24	0.7	182	106	6.6	3.7	0	1	0
54	Siva	40	m	11	2	1	0	0	0	0	0	0	1	0	0	0	0	1	1	0	2	1	1	4	0	0	0	0	0	0	0	0	0	0	0	10	101	23	0.9	160	132	5.8	2.9	1	0	0
55	Munusamy	45	m	7	1	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	5	5	4	22	0	0	0	0	0	0	0	0	0	0	0	10.8	137	32	0.9	129	61	6.6	3.8	0	1	1
56	Muniyan	50	m	12	2	0	0	0	0	0	0	0	1	0	0	1	0	1	1	0	3	3	2	4	0	0	0	0	0	0	0	0	0	0	0	14.4	83	20	0.7	122	106	6.3	3.4	0	1	0
57	Karuppaiya	72	m	13	2	0	0	0	0	0	0	0	1	1	0	1	0	1	1	0	4	3	3	5	0	0	0	0	0	0	0	0	0	0	0	13.6	160	30	1.2	153	90	8.1	4.2	0	1	1
58	Latha	48	f	18	2	0	0	0	0	0	0	0	1	0	0	0	1	0	0	0	4	2	2	8	0	0	0	0	0	0	0	0	0	0	0	6.2	160	24	0.8	148	107	6.8	3.9	0	1	0
59	Subramaniyan	56	m	10	2	0	0	0	0	0	0	0	0	0	0	1	1	0	0	0	3	2	2	3	0	0	0	0	0	0	0	0	0	0	0	14.1	104	18	0.7	135	154	6.8	3.6	0	1	0
60	Basheera Begam	45	f	15	2	0	0	0	1	0	0	0	0	0	0	1	0	0	0	0	4	2	2	7	0	0	0	0	0	0	0	0	0	0	0	10	210	28	0.9	164	158	6.7	3.5	0	1	0
61	Balammal	75	f	7	1	0	0	0	1	0	0	0	0	0	0	0	0	1	0	0	3	3	2	6	0	0	0	0	0	0	0	0	0	0	0	8.4	105	42	1.4	161	168	6.3	3.3	1	0	0
62	Kasi	62	m	14	1	0	1	1	0	0	n	0	0	0	0	0	0	1	1	1	4	4	3	12	0	0	0	0	0	0	0	0	0	0	0	9.2	69	26	0.8	129	101	6.8	4	0	1	1
63	Balakrishnan	55	m	9	2	0	0	0	1	1	0	0	0	0	0	0	0	1	1	1	3	3	3	9	0	0	0	0	0	0	0	0	0	0	0	11.6	109	26	0.8	200	130	6.9	4	0	1	1
64	Senthil	33	m	8	1	0	0	0	1	0	n	0	1	0	0	1	0	1	1	0	5	4	4	12	0	0	0	0	0	0	0	0	0	0	0	13.3	94	24	0.8	170	145	6.7	3.9	0	1	1
65	Anitha	26	f	12	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	5	4	4	13	0	0	0	0	0	0	0	0	0	0	0	9.7	112	22	0.6	205	254	6.5	3.4	1	0	1
66	Kesavan	65	m	15	0	0	1	0	1	0	n	0	0	0	0	0	0	1	0	0	2	2	2	7	0	0	0	0	0	0	0	0	0	0	0	9.4	86	32	1.1	212	170	6	3.2	0	1	0
67	Vignesh	16	m	18	0	0	0	0	1	1	0	1	0	0	0	0	0	0	0	1	4	3	3	3	0	0	0	0	0	0	0	0	0	0	0	12	99	20	0.6	170	145	6.5	4.4	1	0	1
68	Chelladurai	21	m	16	2	0	0	0	1	0	n	0	0	0	0	0	0	0	0	0	4	3	3	10	0	0	0	0	0	0	0	0	0	0	0	13.5	94	28	1	145	135	6.6	3.9	0	1	1
69	Taj	60	m	9	2	0	0	0	0	0	0	0	1	0	0	0	0	1	0	1	3	3	2	4	0	0	0	0	0	0	0	0	0	0	0	12.8	86	34	1.1	175	145	6.3	3.7	0	1	0
70	Mani	53	m	20	0	1	0	0	1	1	0	1	0	1	0	0	0	0	0	0	3	3	2	4	0	0	0	0	0	0	0	0	0	0	0	14.5	202	26	1	204	145	5.7	2.6	1	0	0
71	Gowrinathan	55	m	5	2	0	0	0	1	0	0	0	1	0	0	0	0	0	0	0	3	2	2	4	0	0	0	0	0	0	0	0	0	0	0	12.7	100	30	0.9	213	104	6.1	3.3	0	1	0
72	Thulasiammal	80	f	8	2	0	0	0	1	0	0	0	1	1	0	1	0	0	0	0	4	4	3	9	0	0	0	0	0	0	0	0	0	0	0	14.3	102	22	0.8	155	147	7.1	3	0	1	1
73	Susheela	56	f	7	2	0	0	0	1	0	0	0	1	0	1	0	0	0	0	0	4	3	3	9	0	0	0	0	0	0	0	0	0	0	0	11.2	170	30	1	206	147	6.8	3.2	0	1	1
74	Lakshmanan	56	m	6	1	0	0	0	1	0	0	0	1	0	0	1	0	1	1	0	4	3	3	8	0	0	0	0	0	0	0	0	0	0	0	8.7	99	19	0.9	269	185	5.5	3	0	1	1
75	Annamal	55	f	6	1	1	0	0	0	0	0	0	0	1	1	0	1	0	0	0	3	3	2	6	0	0	0	0	0	0	0	0	0	0	0	10.4	105	61	1.2	170	135	5.5	3	0	1	0
76	Ratnam	63	m	20	0	1	0	0	1	0	0	0	1	0	0	1	0	1	0	0	2	2	1	2	0	0	0	0	0	0	0	0	0	0	0	11.4	110	35	1.2	195	65	6.1	3.3	1	0	0
77	Jayabalan	36	m	13	1	1	0	0	0	0	0	0	1	1	0	0	0	1	0	0	3	3	2	6	0	0	0	0	0	0	0	0	0	0	0	13.9	225	27	0.8	268	192	6.5	3	1	1	0
78	Mani	53	m	20	0	1	0	0	1	1	0	1	0	1	0	1	0	0	0	0	3	3	2	4	0	0	0	0	0	0	0	0	0	0	0	14.5	202	26	1	204	145	5.7	2.6	1	0	0
79	Valarmathi	47	f	6	0	1	0	0	0	0	0	0	1	0	0	1	0	0	0	0	3	3	2	4	0	0	0	0	0	0	0	0	0	0	0	11.5	98	44	1.1	170	135	6.4	3.5	1	0	0
80	Kanniyappan	52	m	15	1	1	0	0	0	0	0	0	0	1	0	0	0	1	0	0	3	3	2	8	0	0	0	0	0	0	0	0	0	0	0	11.2	140	24	0.8	185	128	6.2	3.2	1	0	0
81	Mohd Kasim	46	m	6	2	0	0	0	0	0	0	0	0	0	0	0	0	1	1	0	4	3	2	7	0	0	0	0	0	0	0	0	0	0	0	13	123	30	0.8	169	170	5.1	3	0	1	0
82	Kumar	52	m	14	2	0	0	0	0	0	0	0	1	0	0	1	0	0	0	0	4	4	3	9	0	0	0	0	0	0	0	0	0	0	0	11	160	18	0.6	147	128	6.5	3.2	0	1	1
83	Sugumar	49	m	26	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4	3	3	7	0	0	0	0	0	0	0	0	0	0	0	12.7	129	28	0.9	178	104	6.6	3	0	1	1
84	Perumal	34	m	19	1	0	1	0	0	0	0	0	0	0	0	0	0	1	1	0	4	3	3	12	0	0	0	0	0	0	0	0	0	0	0	14.9	107	15	1	145	118	7.1	4.2	0	1	1

85	Guna	65	f	8	1	0	0	0	1	0	0	0	1	1	0	1	0	0	0	1	4	3	2	9	0	0	0	0	0	0	0	0	0	0	0	10.1	88	17	0.7	185	110	6.8	4	0	1	0
86	Anandhan	45	m	12	2	1	0	0	0	0	0	0	1	0	0	0	1	1	1	0	4	3	3	12	0	0	0	0	0	0	0	0	0	0	0	12.8	90	24	0.7	210	216	6.8	4	1	0	1
87	Kanniyapan	52	m	16	1	1	0	0	0	0	0	0	0	1	0	0	1	0	1	0	4	3	2	8	0	0	0	0	0	0	0	0	0	0	0	16.1	140	24	0.8	198	104	6.2	3.2	0	1	0
88	Poovarasan	20	m	20	1	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	5	4	4	17	0	0	0	0	0	0	0	0	0	0	0	13	99	34	1.2	139	187	6	3.3	0	1	1
89	Loganathan	52	m	18	1	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	4	3	3	9	0	0	0	0	0	0	0	0	0	0	0	15.9	140	27	1	186	132	7	3.9	0	1	1
90	Fathim beevi	60	f	9	1	0	1	0	0	0	0	0	1	1	0	1	1	0	0	0	5	4	3	13	0	0	0	0	0	0	0	0	0	0	0	10	110	28	1.3	192	324	6.7	3.7	0	1	1
91	Prema	38	f	6	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	4	4	3	8	0	0	0	0	0	0	0	0	0	0	0	11	108	33	0.8	175	123	5.9	3.2	0	1	1
92	Chockalingam	65	m	13	1	0	0	0	0	0	0	0	1	0	0	1	1	0	0	0	4	4	3	9	0	0	0	0	0	0	0	0	0	0	0	10.5	112	38	1.2	176	196	6.8	3.3	0	1	1
93	Deivanai	65	f	7	1	0	1	0	0	0	0	0	0	0	0	1	0	0	0	0	4	3	3	10	0	0	0	0	0	0	0	0	0	0	0	9.6	92	22	0.7	182	124	6.9	4.2	0	1	1
94	Elangovan	50	m	13	0	1	0	1	1	1	0	1	0	0	0	0	0	1	1	0	4	4	3	4	0	0	0	0	0	0	0	0	0	0	0	14	97	38	1.1	187	135	6.5	3.5	1	0	1
95	Shajahan Begum	55	f	6	2	0	0	0	0	0	0	0	1	1	0	1	0	0	0	0	4	4	4	9	0	0	0	0	0	0	0	0	0	0	0	11.3	185	40	1.2	247	185	6.5	3	0	1	1
96	Muniyandi	68	m	9	1	0	0	0	1	0	0	0	0	0	0	1	0	1	0	0	4	4	3	12	0	0	0	0	0	0	0	0	0	0	0	7	79	16	1.1	209	176	5.6	3.4	0	1	1
97	Kumari	65	f	9	2	0	0	0	1	0	0	0	1	1	0	0	0	0	0	0	5	4	3	9	0	0	0	0	0	0	0	0	0	0	0	12.4	128	30	1.2	208	165	7	3.2	0	1	1
98	Balakrishnan	56	m	10	2	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	4	4	4	7	0	0	0	0	0	0	0	0	0	0	0	11.6	109	26	0.8	200	130	5.8	3	0	1	1
99	Munusamy	64	m	12	0	0	0	0	1	1	0	1	1	0	0	1	0	1	1	1	4	3	1	4	0	0	0	0	0	0	0	0	0	0	0	12.7	104	24	0.9	128	79	6.3	3.2	1	1	0
100	Kumar	42	m	9	2	0	0	0	0	0	0	0	0	n	0	0	0	0	1	0	4	4	3	9	0	0	0	0	0	0	0	0	0	0	0	13.2	110	34	1.4	202	180	6.8	3.5	0	1	1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Introduction
Stroke is an important cause of disability and second most common cause of death, worldwide. Developing countries account for two thirds of death due to stroke [1, 2]. As per the recent reports, the incidence and thirty day fatality rates is more in India when compared to developed countries[3]. The final outcome of stroke patients has been improved with various strategies like early diagnosis, early prophylactic treatment, early recognition of complications and mobilization[4].
Although the direct effect of brain damage is the main cause for immediate mortality after stroke, the early mortality that occur within the first month after an acute stroke is commonly due to preventable medical complications, that occur either early or late during the course of recovery
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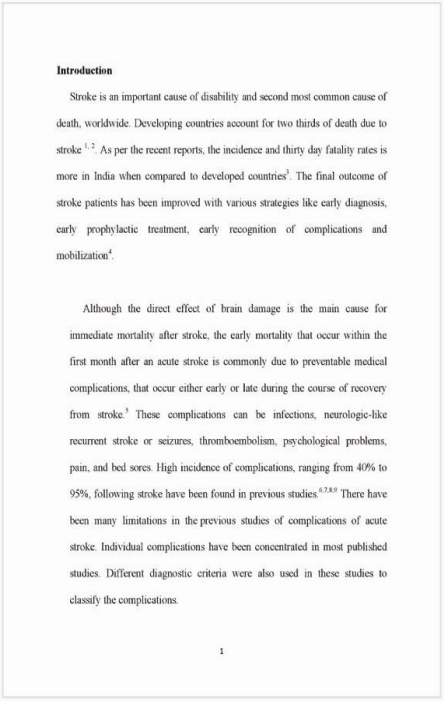

Introduction

Stroke is an important cause of disability and second most common cause of death, worldwide. Developing countries account for two thirds of death due to stroke [1, 2]. As per the recent reports, the incidence and thirty day fatality rates is more in India when compared to developed countries[3]. The final outcome of stroke patients has been improved with various strategies like early diagnosis, early prophylactic treatment, early recognition of complications and mobilization[4].

Although the direct effect of brain damage is the main cause for immediate mortality after stroke, the early mortality that occur within the first month after an acute stroke is commonly due to preventable medical complications, that occur either early or late during the course of recovery from stroke.[5] These complications can be infections, neurologic-like recurrent stroke or seizures, thromboembolism, psychological problems, pain, and bed sores. High incidence of complications, ranging from 40% to 95%, following stroke have been found in previous studies.[6,7,8,9] There have been many limitations in the previous studies of complications of acute stroke. Individual complications have been concentrated in most published studies. Different diagnostic criteria were also used in these studies to classify the complications.

1